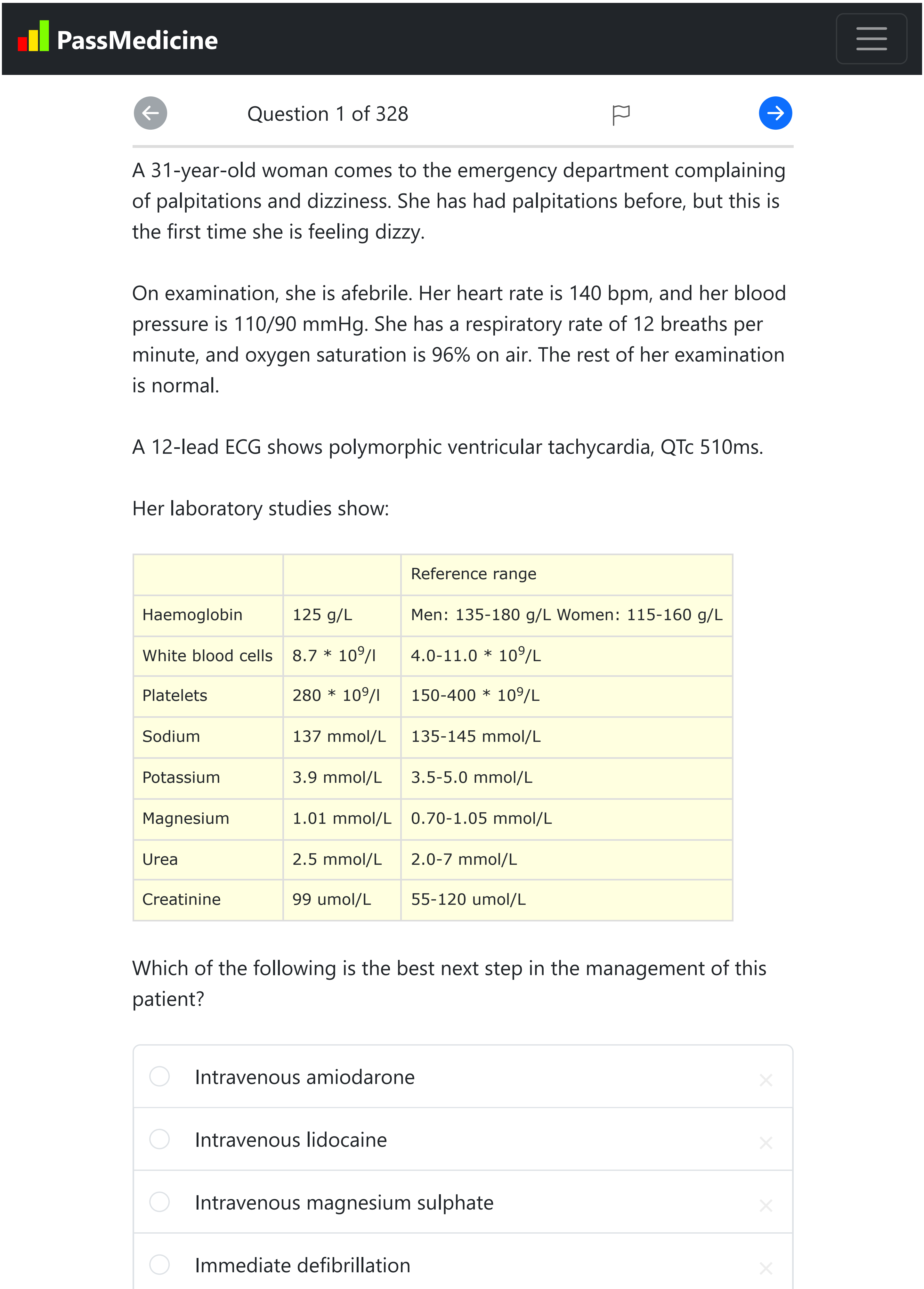
passmedicine PART 2

CARDIOLOGY

2024



A 31-year-old woman comes to the emergency department complaining of palpitations and dizziness. She has had palpitations before, but this is the first time she is feeling dizzy.

On examination, she is afebrile. Her heart rate is 140 bpm, and her blood pressure is 110/90 mmHg. She has a respiratory rate of 12 breaths per minute, and oxygen saturation is 96% on air. The rest of her examination is normal.

A 12-lead ECG shows polymorphic ventricular tachycardia, QTc 510ms.

Her laboratory studies show:

		Reference range
Haemoglobin	125 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	8.7 * 10^9/l	4.0-11.0 * 10^9/L
Platelets	280 * 10^9/l	150-400 * 10^9/L
Sodium	137 mmol/L	135-145 mmol/L
Potassium	3.9 mmol/L	3.5-5.0 mmol/L
Magnesium	1.01 mmol/L	0.70-1.05 mmol/L
Urea	2.5 mmol/L	2.0-7 mmol/L
Creatinine	99 umol/L	55-120 umol/L

Which of the following is the best next step in the management of this patient?

○ Intravenous amiodarone

○ Intravenous lidocaine

○ Intravenous magnesium sulphate

○ Immediate defibrillation

HTTPS://T.ME/+AOWEXXQPZDM1NJZK

○ Synchronised DC cardioversion ×

Submit answer

Reference ranges ⌄

Score: **0%**

1 -

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HTTPS://T.ME/+AOWEXXQPZDM1NJZK

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Creatinine	99 umol/L	55-120 umol/L

Which of the following is the best next step in the management of this patient?

Intravenous amiodarone	13%
Intravenous lidocaine	2%
Intravenous magnesium sulphate	80%
Immediate defibrillation	1%

Synchronised DC cardioversion 4%

IV magnesium sulfate is used to treat torsades de pointes

Important for me Less important

This patient is presenting with an acute episode of torsades de pointes. Her ECG shows polymorphic ventricular tachycardia with a prolonged QT interval, which is consistent with torsades de pointes. It is characterised by rapid, irregular QRS complexes, which appear to be twisting around the ECG baseline. Torsades de pointes is treated with IV magnesium sulphate, even when the serum magnesium levels are normal.

Immediate defibrillation is indicated in patients with pulseless torsades de pointes. This patient is conscious and has a pulse.

Synchronised DC cardioversion is indicated in patients with haemodynamically unstable torsades de pointes. This patient is haemodynamically stable.

IV amiodarone and IV lidocaine can be used to treat monomorphic ventricular tachycardia. Torsades de pointes is a polymorphic ventricular tachycardia.

👍 👎 Discuss (3) Improve

Next question >

Torsades de pointes ★

Torsades de pointes ('twisting of the points') is a form of polymorphic ventricular tachycardia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death.

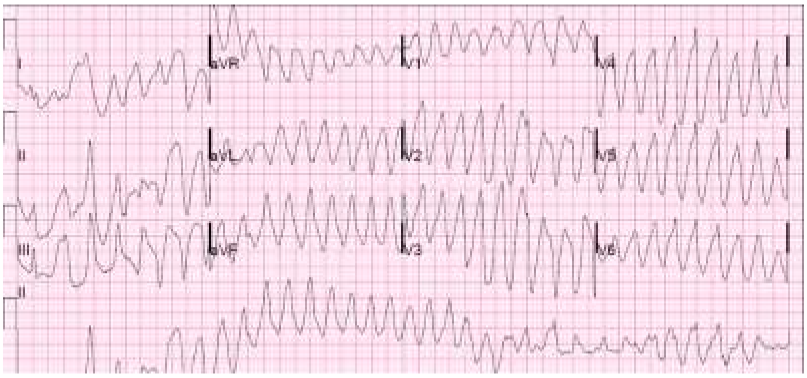
Causes of long QT interval

- congenital
    - Jervell-Lange-Nielsen syndrome
    - Romano-Ward syndrome
- antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- tricyclic antidepressants

- antipsychotics
- chloroquine
- terfenadine
- erythromycin
- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- myocarditis
- hypothermia
- subarachnoid haemorrhage

Management

- IV magnesium sulphate



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ECG showing torsades de pointes

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Textbooks

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Extended textbook

Links

Life in the Fast Lane 👍 19 👎 3

Torsades de pointes

Suggest link Report broken link

Media

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 3 👎 1

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1 ✗

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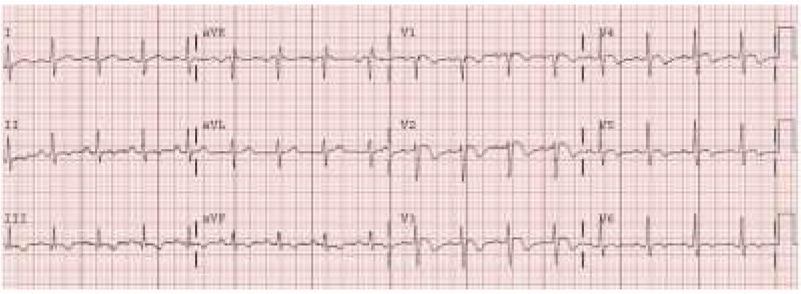
Question 2 of 328

A 35-year-old man presents to his GP with a five day history of dyspnoea, cough, chest pain and palpitations. He describes the chest pain as being worse when he takes a deep breath in and the only relieving factor is ibuprofen. There has been no sputum production or haemoptysis. He has no significant past medical history of note. On examination his pulse is 102/min, blood pressure is 124/78 mmHg and oxygen saturations are 95% on room air. Auscultation of his chest is normal. An ECG is taken:



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What is the most likely diagnosis?

○ Pulmonary embolism

○ Acute coronary syndrome

○ Pericarditis

○ Musculoskeletal chest pain

○ Viral pleurisy

Submit answer

Reference ranges

Score: **0%**

1	✗
2	-

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Question 2 of 328 ✗

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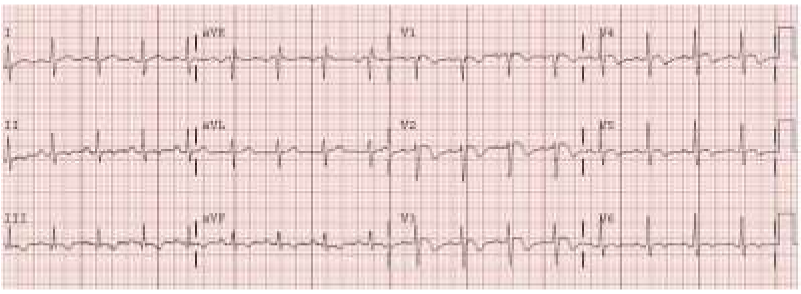

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What is the most likely diagnosis?

Pulmonary embolism	43%
Acute coronary syndrome	5%
Pericarditis	43%
Musculoskeletal chest pain	3%
Viral pleurisy	7%

S1Q3T3 is a classic but uncommon ECG finding in PE

Important for me Less important

The unifying diagnosis here is pulmonary embolism. He has dyspnoea and pleuritic chest pain. The tachycardia and low oxygen saturations are also suggestive. Low oxygen saturations in someone who is otherwise fit and well are unusual and need explaining. Acute coronary syndrome, pericarditis, viral pleurisy and musculoskeletal chest pain would not normally drop your saturations unless complications develop.

The ECG however is the clincher - he has the classic S1Q3T3 changes.

👍 | 👎 | Discuss (15) | Improve

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Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:
- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

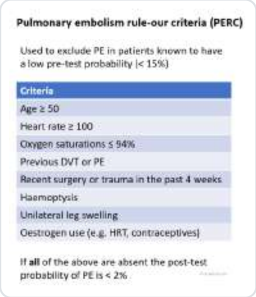
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
    - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5

Clinical feature	**Points**
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores

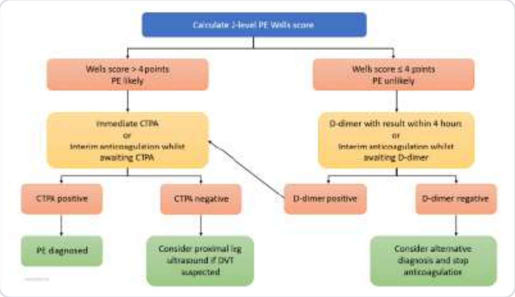
- PE likely - more than 4 points
- PE unlikely - 4 points or less

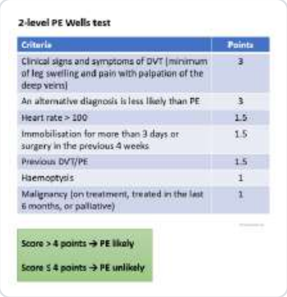
If a PE is 'likely' (more than 4 points)

- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
    - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
    - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points PE likely
Wells score ≤ 4 points PE unlikely
Immediate CTPA or Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours or Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation


2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

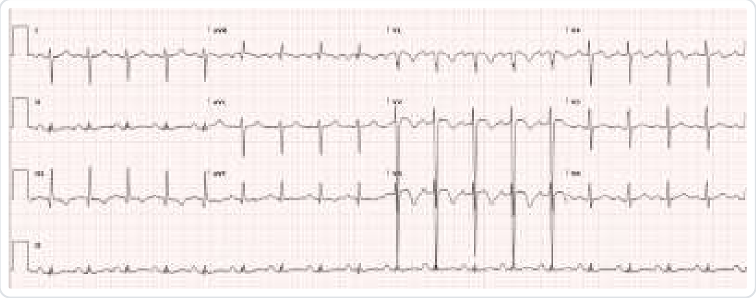
Some other points

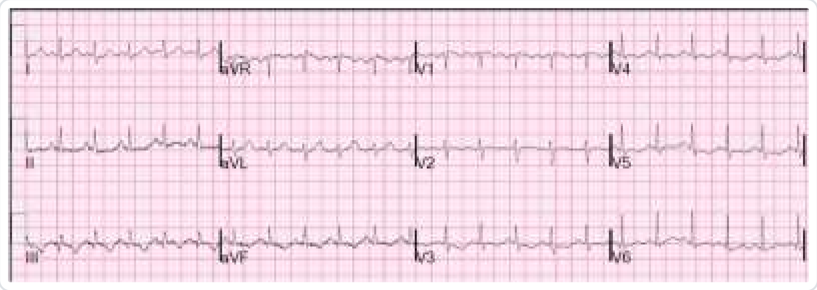
D-dimers

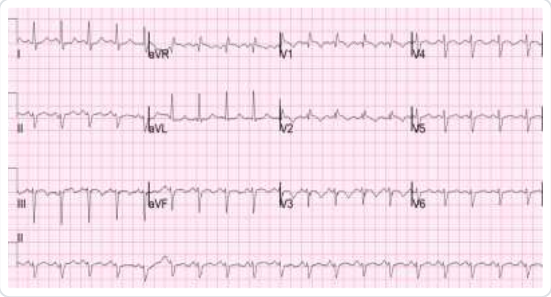
- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen







Chest x-ray

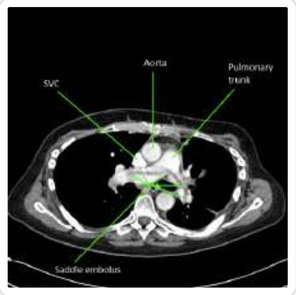
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

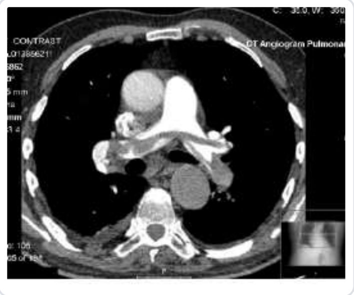
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus


CONTRAST
CT Angiogram Pulmonar

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 13 👎 10

The ECG in Pulmonary Embolism

MDcalc 👍 15 👎 8

Wells' Criteria for Pulmonary Embolism

MDcalc 👍 17 👎 8

PERC Rule for Pulmonary Embolism

NICE 👍 20 👎 9

2020 Venous thromboembolism guidelines

Suggest link Report broken link

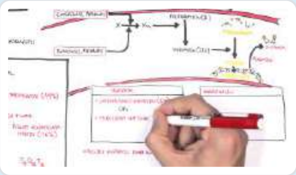
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

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1 ✗

2 ✗

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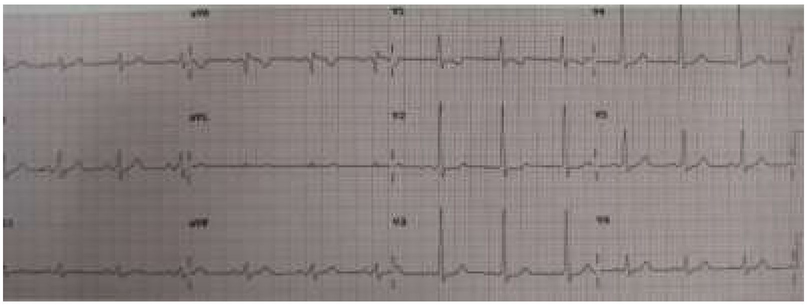
A 26-year-old female presents with a third episode of palpitations associated with shortness of breath and chest discomfort. She has no other past medical history, thyroid function tests unremarkable. She denies taking any recreational drugs and has no significant family history. Her ECG is as follows:



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Which of the following drugs would be safe to administer immediately if she becomes tachycardic?

- ○ Adenosine
- ○ Digoxin
- ○ Diltiazem
- ○ Verapamil
- ○ Procainamide

Submit answer

Reference ranges

Score: **0%**

1 ✗

2 ✗

3 -

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Question 3 of 328 ✗

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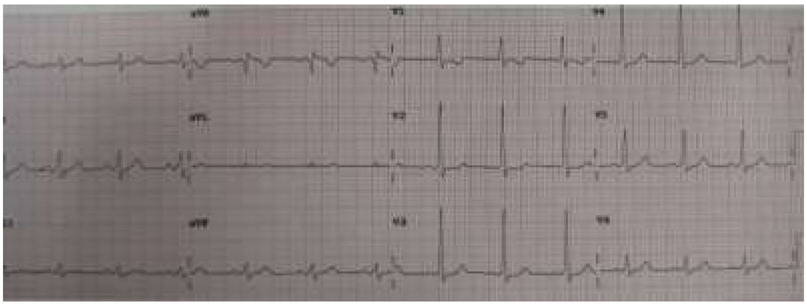

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Which of the following drugs would be safe to administer immediately if she becomes tachycardic?

Adenosine	23%
Digoxin	3%
Diltiazem	5%
Verapamil	11%
Procainamide	57%

The ECG demonstrates Wolff-Parkinson-White (WPW) syndrome, with a delta wave upstroke as a result of pre-excitation prior to the QRS complex. WPW is an atrioventricular re-entry tachycardia, with an accessory pathway allowing rapid conduction between the two channels, bypassing the slow-conducting AV node. Any drug that increases the delay of the normal conduction pathway via the AV node increases conduction via the accessory pathway, potentially inducing ventricular fibrillation or tachycardias. The only drug that does NOT act on the AV node in this setting is procainamide.

Discuss (8) Improve

Next question

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

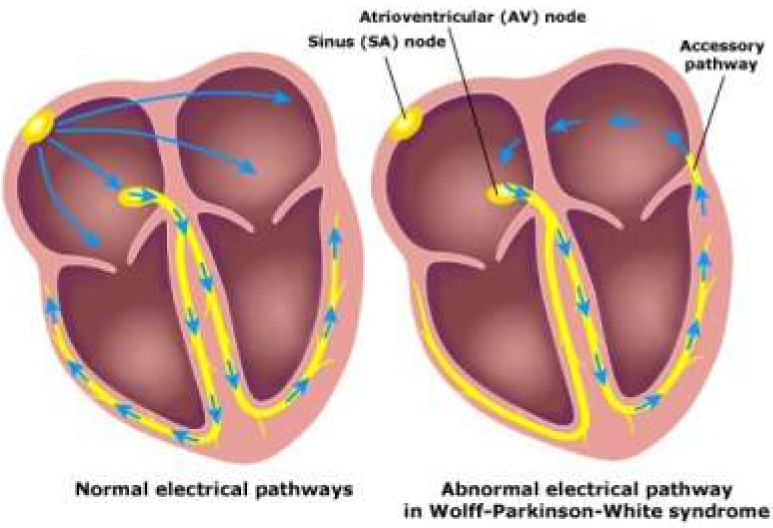
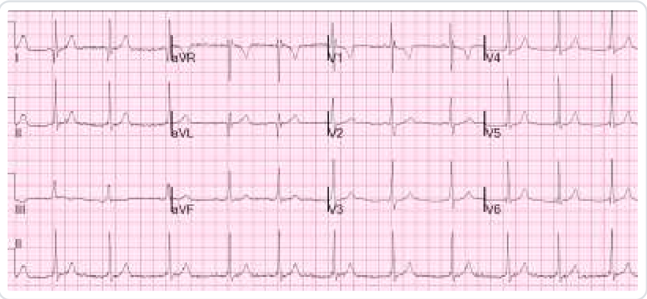
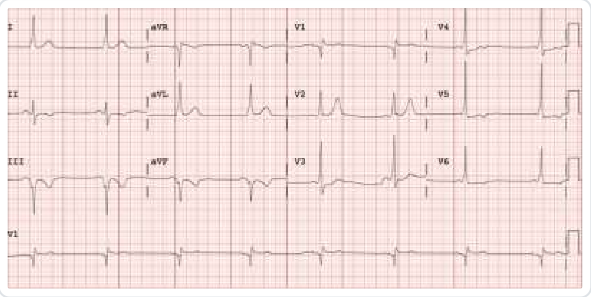

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

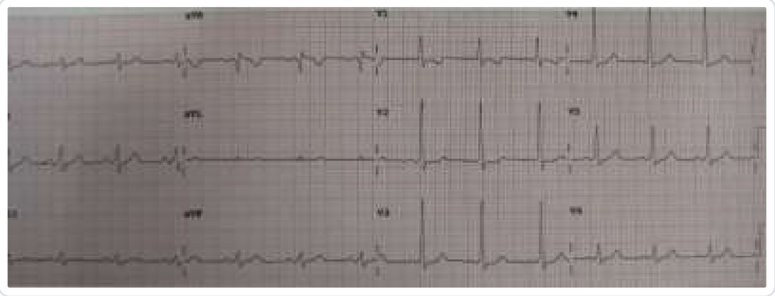
Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
    - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II





Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
  - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

123

Next question

Textbooks

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Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 👍 12 👎 2

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Score: **0%**

1 ✗

2 ✗

3 ✗

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Question 4 of 328

A 54-year-old gentleman is investigated following complaints of frothy urine. Investigations show proteinuria and mild kidney injury, but normal full blood count, liver function tests, inflammatory markers and an ultrasound scan of the kidneys. During examination, he found to have a systolic murmur on the left sternal edge. A transthoracic echocardiogram demonstrates a left ventricular ejection fraction of 55%, normal valves, and increased thickness of the left ventricle to 16mm. In addition, there is a 2mm pericardial effusion and ground-glass changes of the left ventricle. He has no chest pain or shortness of breath. What is the most likely explanation of these echocardiogram findings?

- ○ Cor pulmonale
- ○ Myocarditis
- ○ Mitochondrial disease
- ○ Type 2 diabetes mellitus
- ○ Amyloidosis

Submit answer

Reference ranges

Score: **0%**

1 ✗
2 ✗
3 ✗
4 -

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Question 4 of 328 ✗

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Cor pulmonale	2%
Myocarditis	3%
Mitochondrial disease	2%
Type 2 diabetes mellitus	1%
Amyloidosis	93%

The correct answer is amyloidosis. The symptoms and signs described indicate amyloid deposition in the kidneys and heart. The echocardiogram is particularly telling and the images can be described as a global speckled pattern. Cor pulmonale is unlikely without evidence of respiratory disease, myocarditis would not explain the proteinuria, whilst diabetes would not explain the echocardiogram changes. Mitochondrial disease is more likely with ventricular dilatation but would not show as a ground-glass pattern.

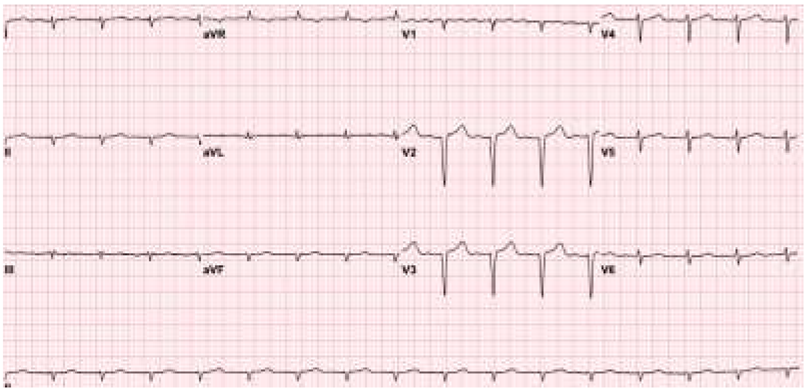
Discuss (2) Improve

Next question ›

Amyloidosis: cardiac ★

Cardiac amyloidosis, also known as amyloid cardiomyopathy, is a rare disorder characterized by the extracellular deposition of insoluble amyloid fibrils in the myocardium, leading to progressive heart failure and conduction abnormalities. Cardiac amyloidosis can occur in isolation or as part of systemic amyloidosis, which affects multiple organs.

Investigations
- ECG: typically shows low-voltage complexes with poor R wave progression in the chest leads (a pseudoinfarction pattern).
- echocardiogram: increased myocardial echogenicity (granular sparkling or 'global speckled' appearance)



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123

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Textbooks

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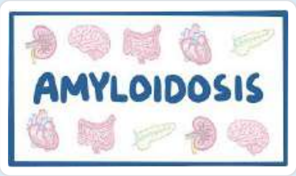
Links

Journal of the American Heart Association 👍 6 👎 0

Updates in Cardiac Amyloidosis: A Review

Suggest link Report broken link

Media



Amyloidosis

Osmosis - YouTube 👍 17 👎 2

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Score: **0%**

1 ✗
2 ✗
3 ✗
4 ✗

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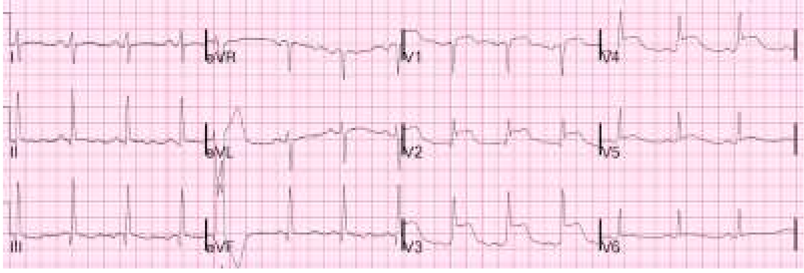
Question 5 of 328

A 57-year-old man presents to the emergency department of a tertiary cardiac centre with a 10-hour history of central chest pain that has not resolved with simple analgesia.

On examination, he is pale and sweaty. His heart rate is 92 beats/min and regular with a blood pressure of 156/84mmHg and saturations of 94% on air. Chest sounds are clear and heart sounds are normal.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the next recommended step in the management of this patient?

○ Apixaban

○ Colchicine

○ Dual antiplatelet therapy and fondaparinux

○ Thrombolysis

○ Transfer to catheter lab for PCI

Submit answer

Reference ranges

Score: **0%**

1	✗
2	✗
3	✗
4	✗
5	-

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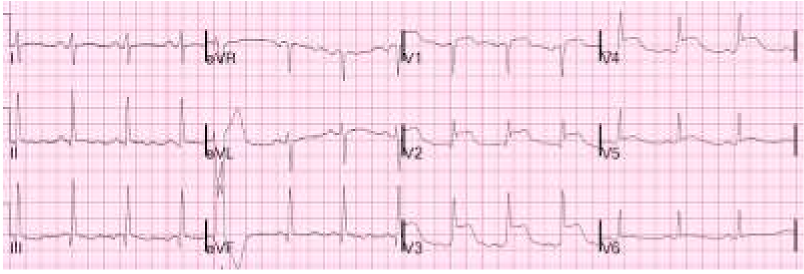
Question 5 of 328 ✓

A 57-year-old man presents to the emergency department of a tertiary cardiac centre with a 10-hour history of central chest pain that has not resolved with simple analgesia.

On examination, he is pale and sweaty. His heart rate is 92 beats/min and regular with a blood pressure of 156/84mmHg and saturations of 94% on air. Chest sounds are clear and heart sounds are normal.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the next recommended step in the management of this patient?

Apixaban	1%
Colchicine	4%
Dual antiplatelet therapy and fondaparinux	13%
Thrombolysis	11%
Transfer to catheter lab for PCI	71%

This patient is having an acute ST-elevation myocardial infarction (STEMI) in the anteroseptal leads (V1-V4). Given the onset of symptoms within 10 hours and the presentation to a centre that is capable of delivering PCI, the most appropriate option would be to **transfer this patient to the catheter lab** immediately.

Apixaban is incorrect. This is used in the treatment of venous thromboembolism or arrhythmias associated with an increased risk for clotting and embolisation (e.g. atrial fibrillation). However, it is not used in the acute management of an ST-elevation myocardial infarction, as this ECG demonstrates.

Colchicine is incorrect. Colchicine is used in the management of pericarditis. Although pericarditis also presents with ST elevation, it is most likely to be widespread and concave in nature. It is also usually associated with PR segment depression, which is not seen here.

Dual antiplatelet therapy and fondaparinux is incorrect. This would be the recommended management for a non-ST elevation myocardial infarction. It is not used in the management of a STEMI.

Thrombolysis is incorrect. Those presenting to the hospital within 12 hours of the onset of chest pain and who can receive PCI within 2 hours, should be offered PCI as the first-line treatment of an acute ischaemic myocardial infarction. If this was not available, then fibrinolysis would be considered.

👍 👎 Discuss (8) Improve

Next question >

Acute coronary syndrome: initial management

★

Acute coronary syndrome (ACS) is a very common and important presentation in medicine. The management of ACS has evolved over recent years, with the development of new drugs and procedures such as percutaneous coronary intervention (PCI).

Emergency departments often have their own protocols based on local factors such as the availability of PCI and hospital drug formularies. The following is based on the 2020 update to the NICE ACS guidelines.

Acute coronary syndrome can be classified as follows:
- ST-elevation myocardial infarction (STEMI): ST-segment elevation + elevated biomarkers of myocardial damage
- non ST-elevation myocardial infarction (NSTEMI): ECG changes but no ST-segment elevation + elevated biomarkers of myocardial

damage
- unstable angina

The management of ACS depends on the particular subtype. NICE management guidance groups the patients into two groups:

- 1. STEMI
- 2. NSTEM/unstable angina

Common management of all patients with ACS

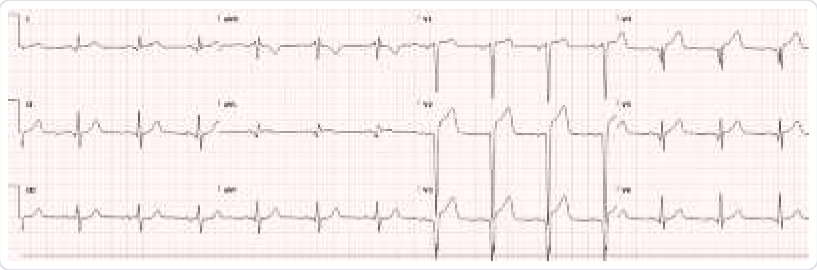
Initial drug therapy

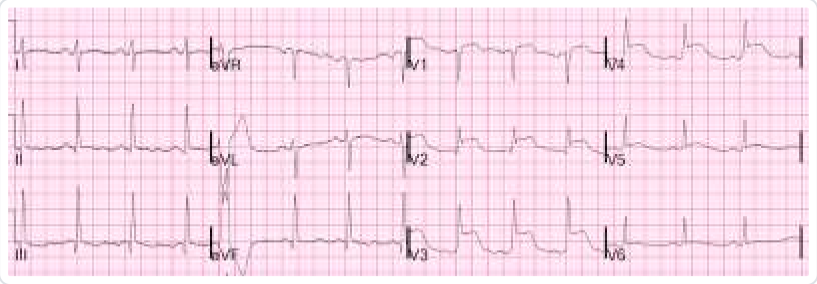
- aspirin 300mg
- oxygen should only be given if the patient has oxygen saturations < 94% in keeping with British Thoracic Society oxygen therapy guidelines
- morphine should only be given for patients with severe pain
    - previously IV morphine was given routinely
    - evidence, however, suggests that this may be associated with adverse outcomes
- nitrates
    - can be given either sublingually or intravenously
    - useful if the patient has ongoing chest pain or hypertension
    - should be used in caution if patient hypotensive

The next step in managing a patient with suspected ACS is to determine whether they meet the ECG criteria for STEMI. It is, of course, important to recognise that these criteria should be interpreted in the context of the clinical history.

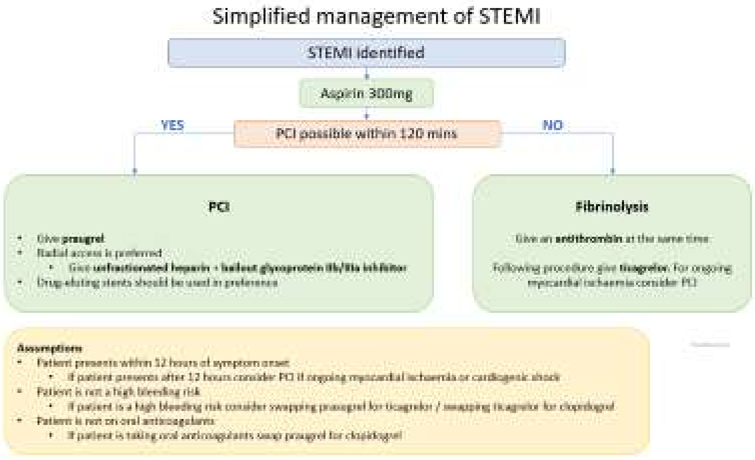
STEMI criteria

- clinical symptoms consistent with ACS (generally of ≥ 20 minutes duration) with persistent (> 20 minutes) ECG features in ≥ 2 contiguous leads of:
    - 2.5 mm (i.e ≥ 2.5 small squares) ST elevation in leads V2-3 in men under 40 years, or ≥ 2.0 mm (i.e ≥ 2 small squares) ST elevation in leads V2-3 in men over 40 years
    - 1.5 mm ST elevation in V2-3 in women
    - 1 mm ST elevation in other leads
    - new LBBB (LBBB should be considered new unless there is evidence otherwise)





Management of STEMI



- Give **prasugrel**
- Radial access is preferred
  - Give **unfractionated heparin + bailout glycoprotein IIb/IIIa inhibitor**
- Drug-eluting stents should be used in preference

Give an **antithrombin** at the same time

Following procedure give **ticagrelor**. For ongoing myocardial ischaemia consider PCI

Assumptions
- Patient presents within 12 hours of symptom onset
  - If patient presents after 12 hours consider PCI if ongoing myocardial ischaemia or cardiogenic shock
- Patient is not a high bleeding risk
  - If patient is a high bleeding risk consider swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
- Patient is not on oral anticoagulants
  - If patient is taking oral anticoagulants swap prasugrel for clopidogrel

Diagram showing the simplified management of STEMI according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

Once a STEMI has been confirmed the first step is to immediately assess eligibility for coronary reperfusion therapy. There are two types of coronary reperfusion therapy:

- percutaneous coronary intervention
  - should be offered if the presentation is within 12 hours of the onset of symptoms AND PCI can be delivered within 120 minutes of the time when fibrinolysis could have been given (i.e. consider fibrinolysis if there is a significant delay in being able to provide PCI)
  - if patients present after 12 hours and still have evidence of ongoing ischaemia then PCI should still be considered

    - drug-eluting stents are now used. Previously 'bare-metal' stents were sometimes used but have higher rates of restenosis
    - radial access is preferred to femoral access
- fibrinolysis
    - should be offered within 12 hours of the onset of symptoms if primary PCI cannot be delivered within 120 minutes of the time when fibrinolysis could have been given
    - a practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) that does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI

If patients are eligible this should be offered as soon as possible.

Percutaneous coronary intervention for patients with STEMI

Further antiplatelet prior to PCI
- this is termed 'dual antiplatelet therapy', i.e. aspirin + another drug
- if the patient is not taking an oral anticoagulant: prasugrel
- if taking an oral anticoagulant: clopidogrel

Drug therapy during PCI
- patients undergoing PCI with radial access:
    - unfractionated heparin with bailout glycoprotein IIb/IIIa inhibitor (GPI) - this is the action of using a GPI during the procedure when it was not intended from the outset, e.g. because of worsening or persistent thrombus
- patients undergoing PCI with femoral access:
    - bivalirudin with bailout GPI

Other procedures during PCI
- thrombus aspiration, but not mechanical thrombus extraction, should be considered
- complete revascularisation should be considered for patients with multivessel coronary artery disease without cardiogenic shock

Fibrinolysis for patients with STEMI

Fibrinolysis used to be the only form of coronary reperfusion therapy available. However, it is used much less commonly now given the widespread availability of PCI.

The contraindications to fibrinolysis and other factors are described in other notes.

Patients undergoing fibrinolysis should also be given an antithrombin drug.

An ECG should be repeated after 60-90 minutes to see if the ECG changes have resolved. If patients have persistent myocardial ischaemia following fibrinolysis then PCI should be considered.

Management of NSTEMI/unstable angina

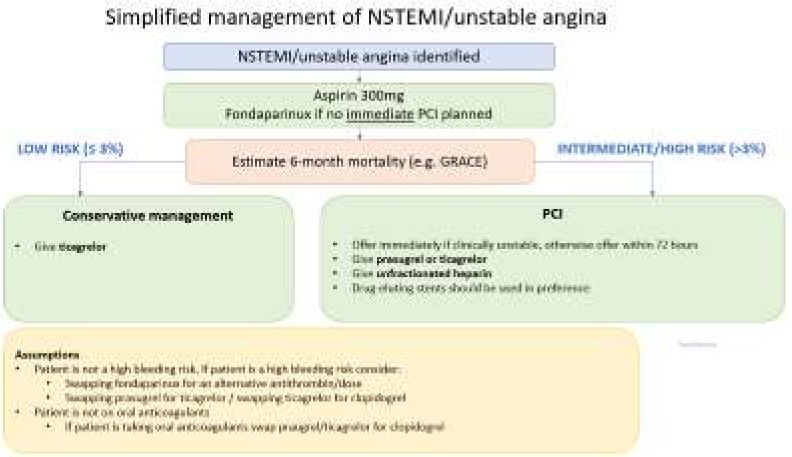

Simplified management of NSTEMI/unstable angina
NSTEMI/unstable angina identified
Aspirin 300mg
Fondaparinux if no immediate PCI planned
LOW RISK (≤ 3%)
Estimate 6-month mortality (e.g. GRACE)
INTERMEDIATE/HIGH RISK (>3%)
Conservative management
Give ticagrelor
PCI
Offer immediately if clinically unstable, otherwise offer within 72 hours
Give prasugrel or ticagrelor
Give unfractionated heparin
Drug-eluting stents should be used in preference
Assumptions
Patient is not a high bleeding risk. If patient is a high bleeding risk consider:
Swapping fondaparinux for an alternative antithrombin/dose
Swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel/ticagrelor for clopidogrel

Diagram showing the simplified management of NSTEMI/unstable angina according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

The management of NSTEMI/unstable angina is complicated and depends on individual patient factors and a risk assessment. The summary below provides an overview but the full NICE guidelines should be reviewed for further details.

Further drug therapy

- antithrombin treatment
  - fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography immediately

    - if immediate angiography is planned or a patients creatinine is > 265 µmol/L then unfractionated heparin should be given

Risk assessment

The Global Registry of Acute Coronary Events (GRACE) is the most widely used tool for risk assessment. It can be calculated using online tools and takes into account the following factors:

- age
- heart rate, blood pressure
- cardiac (Killip class) and renal function (serum creatinine)
- cardiac arrest on presentation
- ECG findings
- troponin levels

This results in the patient being risk stratified as follows:

Predicted 6-month mortality	Risk of future adverse cardiovascular events
1.5% or below	Lowest
> 1.5% to 3.0%	Low
> 3.0% to 6.0%	Intermediate
> 6.0% to 9.0%	High
over 9.0%	Highest

Based on this risk assessment key decisions are made regarding whether a patient has coronary angiography (with follow-on PCI if necessary) or has conservative management. The detailed pros/cons of this decision are covered in other notes.

Which patients with NSTEMI/unstable angina should have coronary angiography (with follow-on PCI if necessary)?

- immediate: patient who are clinically unstable (e.g. hypotensive)
- within 72 hours: patients with a GRACE score > 3% i.e. those at intermediate, high or highest risk
- coronary angiography should also be considered for patients if ischaemia is subsequently experienced after admission

Percutaneous coronary intervention for patients with NSTEMI/unstable angina

Further drug therapy

- unfractionated heparin should be given regardless of whether the patient has had fondaparinux or not
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug) prior to PCI
    - if the patient is not taking an oral anticoagulant: prasugrel or ticagrelor
    - if taking an oral anticoagulant: clopidogrel

Conservative management for patients with NSTEMI/unstable angina

Further drug therapy

- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug)
    - if the patient is not at a high risk of bleeding: ticagrelor
    - if the patient is at a high risk of bleeding: clopidogrel

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 25 👎 28

2016 Acute coronary syndrome guidelines

NICE 👍 64 👎 33

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Media

Acute Coronary Syndrome DETAILED Overview (MI, STEMI, NSTEMI)

Armando Hasudungan - YouTube 👍 5 👎 0

Thrombolytics

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 4 👎 1

Report broken media

Score: **20%**

1	✗
2	✗
3	✗
4	✗
5	✓

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Question 6 of 328

A 54-year-old man attends with recurrent episodes of syncope.

Respiratory rate 18 breaths per minute, SpO2 98% on room air, heart rate 75 beats per minute, BP 114/67 mmHg.

An ECG shows some non-conducted p-waves.

What would be the most appropriate intervention?

- ○ AAI pacemaker
- ○ Ablation
- ○ DDD pacemaker
- ○ Temporary pacing
- ○ VVI pacemaker

Submit answer

Reference ranges

Score: **20%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

6 -

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Question 6 of 328 ✗

A 54-year-old man attends with recurrent episodes of syncope.

Respiratory rate 18 breaths per minute, SpO2 98% on room air, heart rate 75 beats per minute, BP 114/67 mmHg.

An ECG shows some non-conducted p-waves.

What would be the most appropriate intervention?

AAI pacemaker	19%
Ablation	4%
DDD pacemaker	37%
Temporary pacing	6%
VVI pacemaker	34%

In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated

Important for me Less important

The stem describes a stable patient with Mobitz type II AV block. Pacemaker insertion is the most appropriate option.

The most common pacing mode is DDD. This means that there is **d**ual pacing and sensing of both the atrial and ventricles.

DDD pacemaker is the correct option. 'D' stands for dual and means that the pacemaker senses and paces both atrial and ventricular activity. Mobitz type II is a high-degree atrioventricular block, this means that the pacemaker needs to sense both atrial and ventricular activity e.g. if there is a non-conducted p-wave (as in Mobitz type II) the pacemaker senses atrial activity, senses ventricular non-activity, and subsequently stimulates a ventricular response.

VVI pacemaker is incorrect. A VVI pacemaker is a single lead pacemaker that senses and paces the ventricles only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses ventricular activity it inhibits its pacing output. VVI pacemakers, also known as ventricular demand pacemakers, are commonly used for ventricular bradycardia such as atrial fibrillation with a slow ventricular response.

AAI pacemaker is incorrect. An AAI pacemaker is a single lead pacemaker that senses and paces the atria only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses atrial activity it inhibits its pacing output. It is used only for patients with sinus node dysfunction.

Transcutaneous pacing would be used for patients who were unstable as a stop-gap measure whilst they awaited pacemaker insertion. This would not be appropriate in this case as we have a stable and well patient.

Ablation is used to prevent aberrant electrical signalling from causing arrhythmias. In the case of Mobitz type II, the issue is non-conduction rather than aberrant conduction, ablation is therefore not appropriate.

👍 👎 Discuss (6) Improve

Next question ›

Pacemaker: permanent ★

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:

- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):

- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):

- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):

- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)

- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):

- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):

- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:

- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:

- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.

- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

+ Q 123

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Media

DUAL CHAMBER PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **16.7%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗

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A 28-year-old woman attended the emergency department and was subsequently referred to the cardiology team due to concerns about the risk of potential cardiac arrhythmia. The patient presented to the emergency department due to ongoing nausea and vomiting secondary to hyperemesis gravidarum. The patient stated she was 10 weeks pregnant with her first child and had been suffering from these symptoms for the past 6 weeks. Clinical records indicated the patient had also attended the emergency department 3 weeks previously with the same presenting complaint.

On the initial occasion the patient had presented to the emergency department with hyperemesis gravidarum, she had been rehydrated with intravenous fluids and prescribed vitamin B6 and ginger capsules four times daily on the advice of the obstetric team. The patient reported that this treatment had given her only a modest improvement in her symptoms and she had stopped taking these medications after a few days. Subsequently, she had attended an out-of-hours medical centre and been prescribed ondansetron (4 mg three times daily). The patient had found a more significant benefit from ondansetron and had been taking it regularly over the past 2 weeks. In addition to her symptoms of nausea and vomiting, the patient reported feeling tired, lethargic and dehydrated. However, she denied experiencing any other symptoms, including palpitations or pre-syncopal episodes.

The patient's past medical history included a period of intravenous drug use in her early twenties. Three years previously the patient had successfully managed to stop her use of illicit drugs after being enrolled in a methadone treatment program. The patient was now on a consistent methadone dose of 80 mg once daily. There was no other significant past medical history and the patient took no other regular medications.

During the patient's assessment after attending the emergency department, concern was raised over a prolonged corrected QT interval on an ECG performed due to borderline hypokalaemia. The patient's ECG was otherwise unremarkable and was compared to previous ECGs the patient had undergone on her previous visits to the emergency department. Please see below for blood results and data from the patient's current and previous ECG.

Haemoglobin	123 g / dL
Mean cell volume	84.9 fl
White cell count	11.7 x 10>3 / microlitre
Platelets	187 x 10>3 / microlitre
Urea	9.0 mmol / L
Creatinine	82 micromol / L
Sodium	139 mmol / L
Potassium	3.6 mmol / L

Presentation	**Relative date of ECG**	**Corrected QT interval / ms**
2nd ED attendance for hyperemesis	Today	490
1st ED attendance for hyperemesis	3 weeks previously	430
Unrelated ED attendance	4 years previously	415

With regard to the patient's prolonged corrected QT interval, what is the most appropriate strategy for managing her medications?

○ Continue both methadone and ondansetron and admit for cardiac monitoring ×

○ Stop both methadone and ondansetron ×

○ Continue both methadone and ondansetron and arrange outpatient ambulatory ECG ×

○ Continue methadone, stop ondansetron ×

○ Continue ondansetron, stop methadone ×

Submit answer

Reference ranges ﹀

Score: **16.7%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	-

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Question 7 of 328 ✕

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2nd ED attendance for hyperemesis	Today	490
1st ED attendance for hyperemesis	3 weeks previously	430
Unrelated ED attendance	4 years previously	415

With regard to the patient's prolonged corrected QT interval, what is the most appropriate strategy for managing her medications?

- Continue both methadone and ondansetron and admit for cardiac monitoring 5%
- Stop both methadone and ondansetron 30%
- Continue both methadone and ondansetron and arrange outpatient ambulatory ECG 3%
- Continue methadone, stop ondansetron 54%
- Continue ondansetron, stop methadone 8%

The upper limit of normal for corrected QT interval is considered to be 450 ms in men and 460 ms in women. QT prolongation is associated with torsades de pointes (TdP) and sudden cardiac death. However, the presence of a prolonged QT interval does not inevitably lead to TdP and TdP can occur in the presence of a normal QT interval. Each 10 ms increase in QT interval increases the risk of TdP by around 5-7 % and once corrected QT interval exceeds 500 ms, the risk of TdP is markedly increased and intervention is necessary.

The patient is taking 2 medications that are known to prolong the QT interval and these can be seen to have had an additive effect in prolonging the patient's QT interval. Specifically, the corrected QT interval increased from 430 ms when the patient was taking methadone to 490 ms when the patient was taking methadone and ondansetron. The normal baseline ECG suggests that the patient does not have a congenital condition causing QT prolongation. At the time of presentation, the patient's borderline hypokalaemia is a factor increasing the risk of TdP in the setting of her drug-induced prolonged QT interval.

Few recommendations exist for managing the risk of drug-induced prolonged QT interval. Expert advice suggests that when a QT prolonging drug is associated with a corrected QT interval of 480-500 ms in a woman (470-500 ms in a man), or when an increase of 60 ms or greater occurs, dose reduction or discontinuation is advised. If the corrected QT interval exceeds 500 ms then the drug should be discontinued and expert advice sought.

The use of methadone alone does not appear to cause a prolonged QT interval in this patient, whereas her QT interval increased by 60 ms following the addition of ondansetron. Given the importance to the patient's long-term health of adequate opiate replacement therapy, the risks associated with opiate withdrawal to a foetus in early pregnancy and the availability of other anti-emetic medications the most sensible course of action out of the available options is to discontinue ondansetron and continue methadone treatment.

Advice from the cardiology team could be sought to determine if the patient should be admitted for cardiac monitoring. The only available answer to include admission as an option also requires the patient to continue with both medications that prolong the QT interval and so is incorrect. Continuing both medications and discharging the patient for an outpatient ambulatory ECG (presumably to assess for TdP) is not an advisable management plan.

QT interval and drug therapy. BMJ 2016;353:i2732.

Discuss (18) | Improve

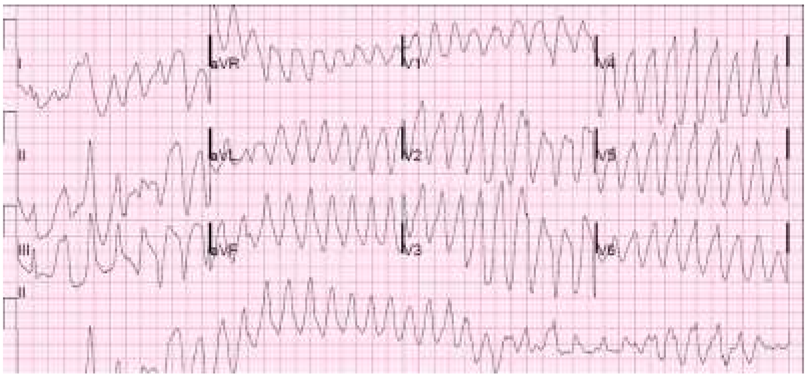
Next question >

Torsades de pointes

Torsades de pointes ('twisting of the points') is a form of polymorphic ventricular tachycardia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death.

Causes of long QT interval
- congenital
    - Jervell-Lange-Nielsen syndrome
    - Romano-Ward syndrome
- antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- tricyclic antidepressants
- antipsychotics
- chloroquine
- terfenadine
- erythromycin
- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- myocarditis
- hypothermia
- subarachnoid haemorrhage

Management
- IV magnesium sulphate



© Image used on license from Dr Smith, University of Minnesota

ECG showing torsades de pointes

123

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Textbooks

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Extended textbook

Links

Life in the Fast Lane 👍 19 👎 3

Torsades de pointes

Suggest link Report broken link

Media

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **14.3%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗

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A 65-year-old man is admitted with central chest pain that has been ongoing for two hours. It radiates to the left arm and is associated with nausea and vomiting. He has a past medical history of hypertension and diabetes which is diet controlled. He takes amlodipine and atorvastatin.

On examination his blood pressure is 147/89 mmHg and his heart rate is 110 beats per minute. His saturations are 96% on room air and he is afebrile His cardiovascular and respiratory examinations are unremarkable.

An ECG shows T wave inversion in leads I, V4, V5 and V6.

Blood tests are as follows:

Hb	131 g/l	Na^+	136 mmol/l
Platelets	$430 * 10^9$/l	K^+	4 mmol/l
WBC	$8 * 10^9$/l	Urea	4 mmol/l
Neuts	$6 * 10^9$/l	Creatinine	84 µmol/l
Lymphs	$1 * 10^9$/l	CRP	41 mg/l
Eosin	$0.3 * 10^9$/l	Trop	130 ng/l

He is diagnosed with non-ST elevation myocardial infarction and commenced on aspirin, clopidogrel and low molecular weight heparin. His pain settles with morphine and glyceryl trinitrate spray within an hour. The following day he is commenced on ramipril and bisoprolol. 3 days later he undergoes an uncomplicated angiogram with stunting of the left circumflex artery.

On systems reviews prior to discharge he comments that he has noticed a rash on his legs and has not been passing much urine despite drinking plenty of water. On examination he has a bluish lacey discolouration over his legs. He has no palpable bladder.

Repeat bloods are as follows:

Hb	12.1 g/l	Na^+	137 mmol/l

Platelets	560 * 10^9/l	K^+	5 mmol/l
WBC	10 * 10^9/l	Urea	12 mmol/l
Neuts	7 * 10^9/l	Creatinine	184 µmol/l
Lymphs	1.2 * 10^9/l	CRP	60 mg/l
Eosin	1.5 * 10^9/l		

What is the most likely cause of his symptoms and deranged blood tests?

○ Cholesterol embolism ×

○ Contrast nephropathy ×

○ Endocarditis ×

○ Hypovolaemia during NSTEMI ×

○ Newly started ACE inhibitor ×

Submit answer

Reference ranges ∨

Score: **14.3%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 -

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Lymphs	1.2 * 10^9/l	CRP	60 mg/l
Eosin	1.5 * 10^9/l		

What is the most likely cause of his symptoms and deranged blood tests?

Cholesterol embolism	91%
Contrast nephropathy	6%
Endocarditis	0%
Hypovolaemia during NSTEMI	1%
Newly started ACE inhibitor	2%

This gentleman as developed an acute kidney injury following angiography. The combination of a lacey rash (livedo reticularis) and raised eosinophils points to a likely diagnosis of cholesterol embolism. Contrast induced nephropathy is also possible following angiography but is not associated with rash or eosinophilia. Although renal function may deteriorate after starting an ACE inhibitor, it is unlikely to cause this degree of derangement so quickly. As the angiogram was uncomplicated, hypovolaemia is unlikely. Endocarditis would be unusual in the absence of fevers and other systemic signs.

Reference - Kronzon et al. Cholesterol Embolization Syndrome. Circulation. 2010:122;631-641.

Discuss (6) | Improve

Next question >

Cholesterol embolisation

Overview

- cholesterol emboli may break off causing renal disease
- the majority of cases are secondary to vascular surgery or angiography. Other causes include severe atherosclerosis, particularly in large arteries such as the aorta

Features

- eosinophilia
- purpura
- renal failure
- livedo reticularis

+ Q 123

Next question >

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Score: **12.5%**

1	✗
2	✗
3	✗
4	✗
5	✓

6	✗
7	✗
8	✗

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Question 9 of 328

A 52-year-old man is asymptomatic but concerned about his risk of having a heart attack. His 58-year-old brother died of a myocardial infarction 2 months ago. He has a past medical history of hypertension, for which he takes perindopril and amlodipine. His sister takes tablets for diabetes and high blood pressure. He successfully quit smoking over 1 year ago and drinks approximately 2 units of alcohol per day.

On examination, his blood pressure is 130/70 mmHg and BMI is 32 kg/m2.

Investigations:

haemoglobin	16g/dL (13-18)
mean corpuscular volume	90 fL (80-96)
serum cholesterol	5.0 mmol/L (<5.2)
serum LDL cholesterol	3.1 mmol/L (<3.36)
serum HDL cholesterol	1.55 mmol/L (>1.55)
fasting serum triglycerides	1.6 mmol/L (0.451.69)
haemoglobin A1c	5.4% (4.06.0)
serum aspartate aminotransferase	19 U/L (131)
plasma thyroid-stimulating hormone	2.4 mU/L (0.45.0)
estimated glomerular filtration rate (MDRD)	85 mL/min (>60)

His QRISK2 score estimates a 13% 10 year risk of myocardial infarction or stroke.

In addition to discussing lifestyle modifications, what treatment should you offer this patient?

○ Aspirin

○ Atorvastatin

○ Indapamide ×

○ Metformin ×

○ Omega-3-polyunsaturated fatty acid supplements ×

Submit answer

Reference ranges ∨

Score: **12.5%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	-

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A 52-year-old man is asymptomatic but concerned about his risk of having a heart attack. His 58-year-old brother died of a myocardial infarction 2 months ago. He has a past medical history of hypertension, for which he takes perindopril and amlodipine. His sister takes tablets for diabetes and high blood pressure. He successfully quit smoking over 1 year ago and drinks approximately 2 units of alcohol per day.

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plasma thyroid-stimulating hormone	2.4 mU/L (0.45.0)
estimated glomerular filtration rate (MDRD)	85 mL/min (>60)

His QRISK2 score estimates a 13% 10 year risk of myocardial infarction or stroke.

In addition to discussing lifestyle modifications, what treatment should you offer this patient?

Aspirin	8%
Atorvastatin	84%

Indapamide	1%
Metformin	3%
Omega-3-polyunsaturated fatty acid supplements	4%

The 2014 NICE guidelines: Cardiovascular disease: risk assessment and reduction, including lipid modification have lowered the UK threshold for offering a statin for primary prevention to a 10% or greater 10year risk of developing cardiovascular disease, according to the QRISK2 score. The previous recommendation advised offering a statin to patients with a 20% 10-year risk. A high-intensity low cost statin should be 1st line in this context, such as atorvastatin 20mg.

Prescribing aspirin for primary prevention is controversial, whether the benefits in primary prevention outweigh the bleeding risks is unclear, therefore most guidelines do not recommend aspirin in primary prevention. Unlike in secondary prevention where the benefits usually outweigh the risks.

The NICE guidelines advise NOT prescribing omega-3-fatty acid supplements for primary or secondary prevention of cardiovascular disease, as they have not shown to be efficacious in reducing cardiovascular risk in meta-analyses. However, guidance continues to encourage consuming regular portions of oily fish, the 2014 nice guidance specifically advises: 2 portions of fish per week, including a portion of oily fish.

Metformin would be inappropriate in this patient as he is not diabetic.

This patients hypertension is well controlled, therefore indapamide would be inappropriate.

Sources:

NICE guidelines Cardiovascular disease: risk assessment and reduction, including lipid modification:
http://www.nice.org.uk/guidance/cg181/chapter/1-recommendations

Meta-analysis in JAMA re: omega-3 supplements:
http://jama.jamanetwork.com/article.aspx?articleid=1357266

NICE guidlines Omega-3 fatty acid supplements:
https://www.nice.org.uk/advice/ktt4/chapter/evidence-context

Dont use aspirin for primary prevention of cardiovascular disease:
http://www.bmj.com/content/340/bmj.c1805

👍 👎 💬 Discuss (2) Improve

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Statins ★

Statins inhibit the action of HMG-CoA reductase, the rate-limiting enzyme in hepatic cholesterol synthesis.

Adverse effects

- myopathy: includes myalgia, myositis, rhabdomyolysis and asymptomatic raised creatine kinase. Risks factors for myopathy include advanced age, female sex, low body mass index and presence of multisystem disease such as diabetes mellitus. Myopathy is more common in lipophilic statins (simvastatin, atorvastatin) than relatively hydrophilic statins (rosuvastatin, pravastatin, fluvastatin)
- liver impairment: the 2014 NICE guidelines recommend checking LFTs at baseline, 3 months and 12 months. Treatment should be discontinued if serum transaminase concentrations rise to and persist at 3 times the upper limit of the reference range
- there is some evidence that statins may increase the risk of intracerebral haemorrhage in patients who've previously had a stroke. This effect is not seen in primary prevention. For this reason the Royal College of Physicians recommend avoiding statins in patients with a history of intracerebral haemorrhage

Contraindications

- macrolides (e.g. erythromycin, clarithromycin) are an important interaction. Statins should be stopped until patients complete the course
- pregnancy

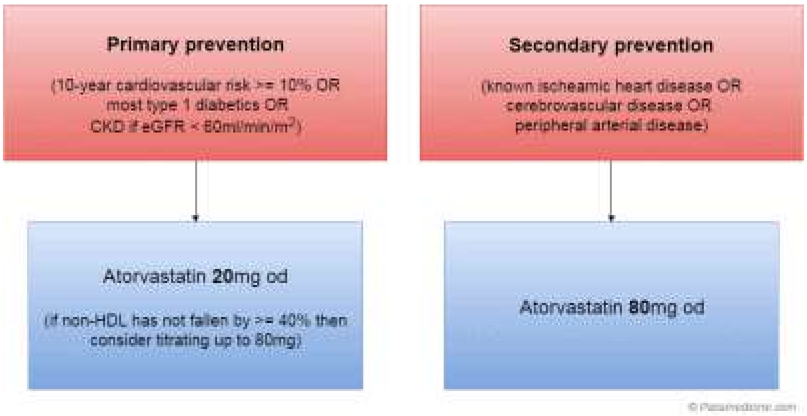
Who should receive a statin?

- all people with established cardiovascular disease (stroke, TIA, ischaemic heart disease, peripheral arterial disease)
- following the 2014 update, NICE recommend anyone with a 10-year cardiovascular risk >= 10%
- patients with type 2 diabetes mellitus should now be assessed using QRISK2 like other patients are, to determine whether they should be started on statins
- patients with type 1 diabetes mellitus who were diagnosed more than 10 years ago OR are aged over 40 OR have established nephropathy

Statins should be taken at night as this is when the majority of cholesterol synthesis takes place. This is especially true for simvastatin which has a shorter half-life than other statins.

NICE currently recommends the following for the prevention of cardiovascular disease::

- atorvastatin 20mg for primary prevention
    - increase the dose if non-HDL has not reduced for >= 40%
- atorvastatin 80mg for secondary prevention


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od
© Passmedicine.com

Graphic showing choice of statin.

123

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Links

RCP 👍 15 👎 21

2016 Stroke guidelines

NICE 👍 20 👎 16

2014 Lipid modification guidelines

Clinical Knowledge Summaries 👍 18 👎 14

Lipids management

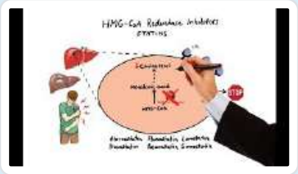
NICE 👍 18 👎 12

2022 Type 2 diabetes guidelines

Suggest link

Report broken link

Media


HMG-CoA Reductase Inhibitors
STATINS

Drugs for hyperlipidaemia

Speed Pharmacology - YouTube 👍 3 👎 0

Report broken media

Score: **11.1%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗

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Question 10 of 328

A 60-year-old man presents to the emergency department with crushing central chest pain. The pain came on suddenly approximately one hour ago. A 12-lead ECG reveals >2mm of ST elevation in all anterior and lateral leads. The nearest centre offering primary angioplasty is 140 minutes away. There is no contraindication to thrombolysis.

In addition to standard acute coronary syndrome treatment (analgesia, aspirin, a heparin and a P2Y12 inhibitor), what is the best management for this patient?

- ○ Immediate transfer for acute angioplasty ×
- ○ Non-urgent angioplasty ×
- ○ Thrombolysis ×
- ○ Thrombolysis and immediate transfer for acute angioplasty ×
- ○ Thrombolysis and non-urgent angioplasty ×

Submit answer

Reference ranges

Score: **11.1%**

1 ✗
2 ✗
3 ✗
4 ✗

5	✓
6	✗
7	✗
8	✗
9	✗
10	-

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In addition to standard acute coronary syndrome treatment (analgesia, aspirin, a heparin and a P2Y12 inhibitor), what is the best management for this patient?

Immediate transfer for acute angioplasty	10%
Non-urgent angioplasty	0%
Thrombolysis	18%
🟩 Thrombolysis and immediate transfer for acute angioplasty	57%
🟥 Thrombolysis and non-urgent angioplasty	15%

The recommended door-to-balloon aka door-to-device time for primary percutaneous coronary intervention (PCI) is within 90 minutes. However, many centres do not have provision for primary PCI and are unable to transfer patients to a centre that does in a timely manner.

The NORDISTEMI TRIAL (NORwegian study on DIstrict treatment of ST-Elevation Myocardial Infarction) demonstrated that in centres with a long transfer time (mean transfer time was 130 minutes in this trial) for PCI, patients with STEMI should initially receive thrombolysis, with subsequent immediate transfer for PCI. For patients with STEMI this approach reduced the rate of death, re-infarction, or stroke at 12 months compared to patients treated with a conservative, ischaemia-guided approach (e.g thrombolysis alone or thrombolysis with rescue angioplasty).

Sources:

Bohmer, E., et al., Efficacy and safety of immediate angioplasty versus ischemia-guided management after thrombolysis in acute myocardial infarction in areas with very long transfer distances results of the NORDISTEMI (NORwegian study on DIstrict treatment of ST-elevation myocardial infarction). J Am Coll Cardiol, 2010. 55(2): p. 102-10. http://www.sciencedirect.com/science/article/pii/S0735109709026424

2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction
http://circ.ahajournals.org/content/127/4/e362.full#F2

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Next question >

Acute coronary syndrome: initial management

★

Acute coronary syndrome (ACS) is a very common and important presentation in medicine. The management of ACS has evolved over recent years, with the development of new drugs and procedures such as percutaneous coronary intervention (PCI).

Emergency departments often have their own protocols based on local factors such as the availability of PCI and hospital drug formularies. The following is based on the 2020 update to the NICE ACS guidelines.

Acute coronary syndrome can be classified as follows:

- ST-elevation myocardial infarction (STEMI): ST-segment elevation + elevated biomarkers of myocardial damage
- non ST-elevation myocardial infarction (NSTEMI): ECG changes but no ST-segment elevation + elevated biomarkers of myocardial damage
- unstable angina

The management of ACS depends on the particular subtype. NICE management guidance groups the patients into two groups:

- 1. STEMI
- 2. NSTEM/unstable angina

Common management of all patients with ACS

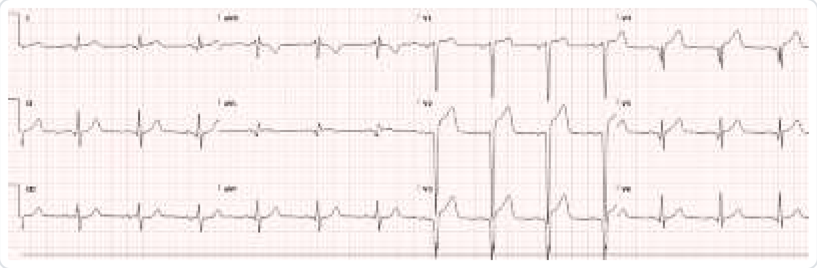
Initial drug therapy

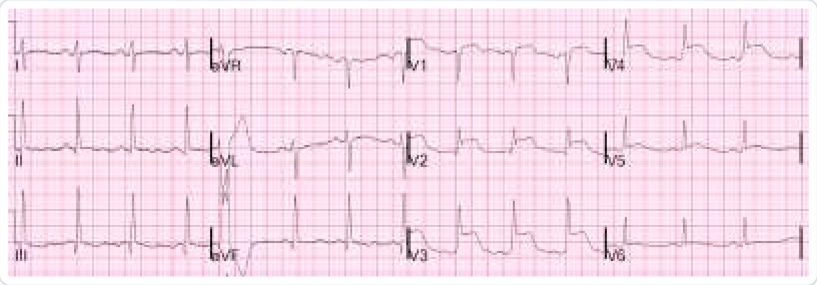
- aspirin 300mg
- oxygen should only be given if the patient has oxygen saturations < 94% in keeping with British Thoracic Society oxygen therapy guidelines
- morphine should only be given for patients with severe pain
  - previously IV morphine was given routinely
  - evidence, however, suggests that this may be associated with adverse outcomes
- nitrates
  - can be given either sublingually or intravenously
  - useful if the patient has ongoing chest pain or hypertension
  - should be used in caution if patient hypotensive

The next step in managing a patient with suspected ACS is to determine whether they meet the ECG criteria for STEMI. It is, of course, important to recognise that these criteria should be interpreted in the context of the clinical history.

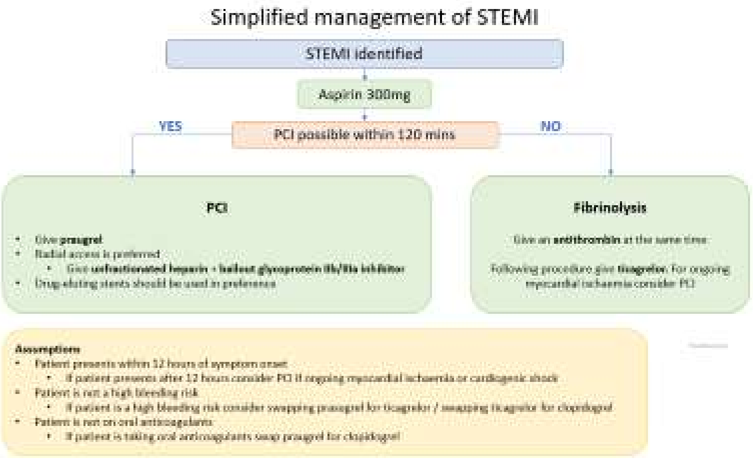
STEMI criteria

- clinical symptoms consistent with ACS (generally of ≥ 20 minutes duration) with persistent (> 20 minutes) ECG features in ≥ 2 contiguous leads of:
  - 2.5 mm (i.e ≥ 2.5 small squares) ST elevation in leads V2-3 in men under 40 years, or ≥ 2.0 mm (i.e ≥ 2 small squares) ST elevation in leads V2-3 in men over 40 years
  - 1.5 mm ST elevation in V2-3 in women
  - 1 mm ST elevation in other leads
  - new LBBB (LBBB should be considered new unless there is evidence otherwise)





Management of STEMI



- Give **prasugrel**
- Radial access is preferred
  - Give **unfractionated heparin + bailout glycoprotein IIb/IIIa inhibitor**
- Drug-eluting stents should be used in preference

Give an **antithrombin** at the same time

Following procedure give **ticagrelor**. For ongoing myocardial ischaemia consider PCI

Assumptions
- Patient presents within 12 hours of symptom onset
  - If patient presents after 12 hours consider PCI if ongoing myocardial ischaemia or cardiogenic shock
- Patient is not a high bleeding risk
  - If patient is a high bleeding risk consider swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
- Patient is not on oral anticoagulants
  - If patient is taking oral anticoagulants swap prasugrel for clopidogrel

Diagram showing the simplified management of STEMI according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

Once a STEMI has been confirmed the first step is to immediately assess eligibility for coronary reperfusion therapy. There are two types of coronary reperfusion therapy:

- percutaneous coronary intervention
  - should be offered if the presentation is within 12 hours of the onset of symptoms AND PCI can be delivered within 120 minutes of the time when fibrinolysis could have been given (i.e. consider fibrinolysis if there is a significant delay in being able to provide PCI)
  - if patients present after 12 hours and still have evidence of ongoing ischaemia then PCI should still be considered

    - drug-eluting stents are now used. Previously 'bare-metal' stents were sometimes used but have higher rates of restenosis
    - radial access is preferred to femoral access
- fibrinolysis
    - should be offered within 12 hours of the onset of symptoms if primary PCI cannot be delivered within 120 minutes of the time when fibrinolysis could have been given
    - a practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) that does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI

If patients are eligible this should be offered as soon as possible.

Percutaneous coronary intervention for patients with STEMI

Further antiplatelet prior to PCI
- this is termed 'dual antiplatelet therapy', i.e. aspirin + another drug
- if the patient is not taking an oral anticoagulant: prasugrel
- if taking an oral anticoagulant: clopidogrel

Drug therapy during PCI
- patients undergoing PCI with radial access:
    - unfractionated heparin with bailout glycoprotein IIb/IIIa inhibitor (GPI) - this is the action of using a GPI during the procedure when it was not intended from the outset, e.g. because of worsening or persistent thrombus
- patients undergoing PCI with femoral access:
    - bivalirudin with bailout GPI

Other procedures during PCI
- thrombus aspiration, but not mechanical thrombus extraction, should be considered
- complete revascularisation should be considered for patients with multivessel coronary artery disease without cardiogenic shock

Fibrinolysis for patients with STEMI

Fibrinolysis used to be the only form of coronary reperfusion therapy available. However, it is used much less commonly now given the widespread availability of PCI.

The contraindications to fibrinolysis and other factors are described in other notes.

Patients undergoing fibrinolysis should also be given an antithrombin drug.

An ECG should be repeated after 60-90 minutes to see if the ECG changes have resolved. If patients have persistent myocardial ischaemia following fibrinolysis then PCI should be considered.

Management of NSTEMI/unstable angina

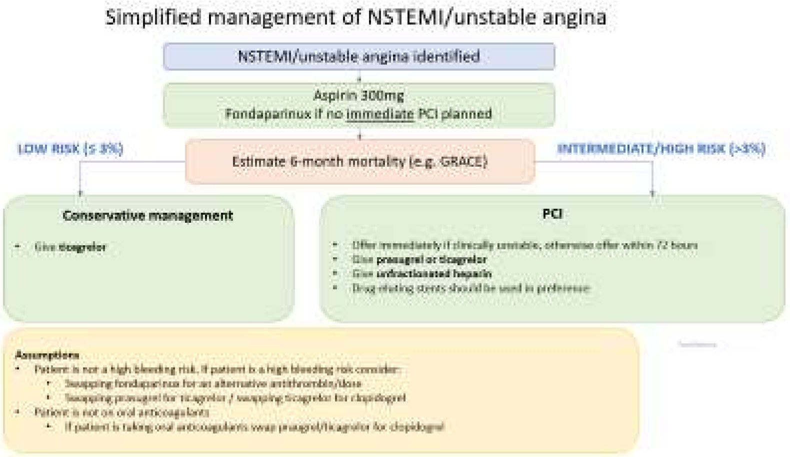

Simplified management of NSTEMI/unstable angina
NSTEMI/unstable angina identified
Aspirin 300mg
Fondaparinux if no immediate PCI planned
LOW RISK (≤ 3%)
Estimate 6-month mortality (e.g. GRACE)
INTERMEDIATE/HIGH RISK (>3%)
Conservative management
Give ticagrelor
PCI
Offer immediately if clinically unstable, otherwise offer within 72 hours
Give prasugrel or ticagrelor
Give unfractionated heparin
Drug-eluting stents should be used in preference
Assumptions
Patient is not a high bleeding risk. If patient is a high bleeding risk consider:
Swapping fondaparinux for an alternative antithrombin/dose
Swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel/ticagrelor for clopidogrel

Diagram showing the simplified management of NSTEMI/unstable angina according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

The management of NSTEMI/unstable angina is complicated and depends on individual patient factors and a risk assessment. The summary below provides an overview but the full NICE guidelines should be reviewed for further details.

Further drug therapy

- antithrombin treatment
  - fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography immediately

    - if immediate angiography is planned or a patients creatinine is > 265 μmol/L then unfractionated heparin should be given

Risk assessment

The Global Registry of Acute Coronary Events (GRACE) is the most widely used tool for risk assessment. It can be calculated using online tools and takes into account the following factors:

- age
- heart rate, blood pressure
- cardiac (Killip class) and renal function (serum creatinine)
- cardiac arrest on presentation
- ECG findings
- troponin levels

This results in the patient being risk stratified as follows:

Predicted 6-month mortality	Risk of future adverse cardiovascular events
1.5% or below	Lowest
> 1.5% to 3.0%	Low
> 3.0% to 6.0%	Intermediate
> 6.0% to 9.0%	High
over 9.0%	Highest

Based on this risk assessment key decisions are made regarding whether a patient has coronary angiography (with follow-on PCI if necessary) or has conservative management. The detailed pros/cons of this decision are covered in other notes.

Which patients with NSTEMI/unstable angina should have coronary angiography (with follow-on PCI if necessary)?

- immediate: patient who are clinically unstable (e.g. hypotensive)
- within 72 hours: patients with a GRACE score > 3% i.e. those at intermediate, high or highest risk
- coronary angiography should also be considered for patients if ischaemia is subsequently experienced after admission

Percutaneous coronary intervention for patients with NSTEMI/unstable angina

Further drug therapy

- unfractionated heparin should be given regardless of whether the patient has had fondaparinux or not
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug) prior to PCI
    - if the patient is not taking an oral anticoagulant: prasugrel or ticagrelor
    - if taking an oral anticoagulant: clopidogrel

Conservative management for patients with NSTEMI/unstable angina

Further drug therapy

- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug)
    - if the patient is not at a high risk of bleeding: ticagrelor
    - if the patient is at a high risk of bleeding: clopidogrel

123

Next question >

Textbooks

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Links

SIGN 👍 25 👎 28

2016 Acute coronary syndrome guidelines

NICE 👍 64 👎 33

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Media

Acute Coronary Syndrome DETAILED Overview (MI, STEMI, NSTEMI)

Armando Hasudungan - YouTube 👍 5 👎 0

Thrombolytics

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 4 👎 1

Report broken media

Score: **10%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗

7	✗
8	✗
9	✗
10	✗

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Question 11 of 328

A 73-year-old gentleman sees his GP with progressive shortness of breath. He can now walk 10 yards before stopping to catch his breath and he is unable to lie flat. He sleeps upright in his armchair and has stopped sleeping in his bed over the last three months.

He has known hypertension and has had two previous myocardial infarctions. On examination, his apex beat is displaced to the 7th intercostal space in the left mid-axillary line and there are crepitations in the two lung bases. His JVP is raised at 5cm and there is bilateral leg oedema.

Troponin	<20 ng/ml
B-natriuretic peptide	14,000 ng/L (normal range 0-200)
Chest X-ray	cardiomegaly and increased alveolar shadowing in the perihilar areas
ECG	QRS 110ms, large volume complexes in chest leads

Which intervention is proven to reduce mortality in this case?

○ Cardiac Resynchronisation Therapy (CRT)

○ Digoxin

○ Furosemide

○ Ramipril

○ Coronary artery bypass graft

Submit answer

Reference ranges

Question 11 of 328 ✗

A 73-year-old gentleman sees his GP with progressive shortness of breath. He can now walk 10 yards before stopping to catch his breath and he is unable to lie flat. He sleeps upright in his armchair and has stopped sleeping in his bed over the last three months.

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Chest X-ray	cardiomegaly and increased alveolar shadowing in the perihilar areas
ECG	QRS 110ms, large volume complexes in chest leads

Which intervention is proven to reduce mortality in this case?

Cardiac Resynchronisation Therapy (CRT)	27%
Digoxin	1%
Furosemide	8%
Ramipril	62%
Coronary artery bypass graft	2%

ACE inhibitors offer prognostic benefit in chronic heart failure

Important for me Less important

Of the above options, ramipril is proven to reduce mortality in congestive cardiac failure through adjusting remodelling of the heart.

Digoxin and furosemide can help symptoms but no trial shows a benefit in mortality. CRT can be helpful in cases of ventricular dyssynchrony but the QRS is narrow and this gentleman would not benefit from it. Coronary artery bypass grafting may be useful if this gentleman had symptoms of angina but he does not.

👍 | 👎 | Discuss (2) | Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

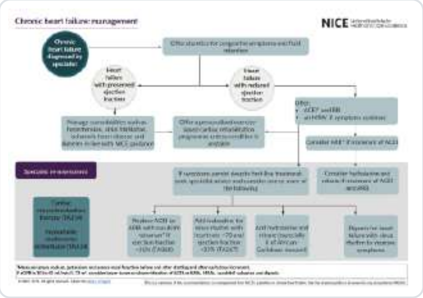
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster

every 5 years


Chronic heart failure: management
NICE

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

2012 Guidance on ivabradine

NICE 42 15

2018 Chronic heart failure guidelines

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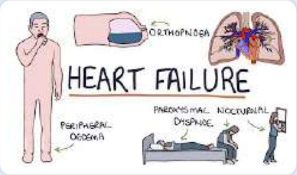
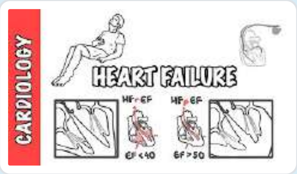
Media


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOE
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1

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Heart failure

Osmosis - YouTube 👍 0 👎 2

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Score: **9.1%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗
10	✗

11 ✗

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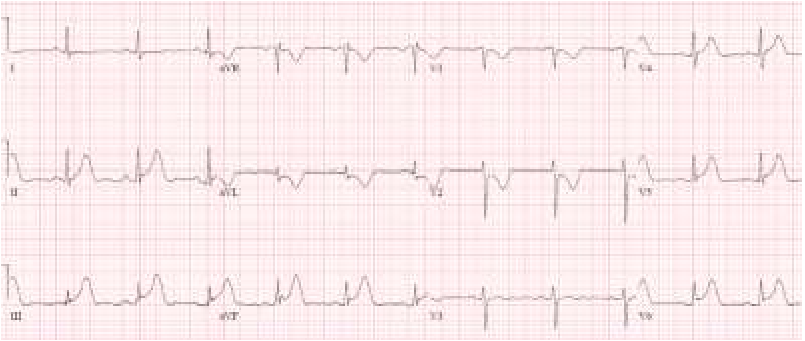
Question 12 of 328

A 75-year-old male presents to the emergency department with central crushing chest pain which has been ongoing for 4 hours. It is associated with shortness of breath.

Blood results are as follows:

Hb	125 g/L	Male: (135-180) Female: (115 - 160)
Platelets	424 * 10^9/L	(150 - 400)
WBC	14.6 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	134 µmol/L	(55 - 120)
Troponin	8422 ng/L	(< 15)

An ECG is performed:



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What is the most likely diagnosis?

○ Anterolateral STEMI ×

○ Inferolateral STEMI ×

○ Myocarditis ×

○ Pericarditis ×

○ Posterior STEMI ×

Submit answer

Reference ranges ∨

Score: **9.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 -

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Na^+	135 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	134 µmol/L	(55 - 120)
Troponin	8422 ng/L	(< 15)

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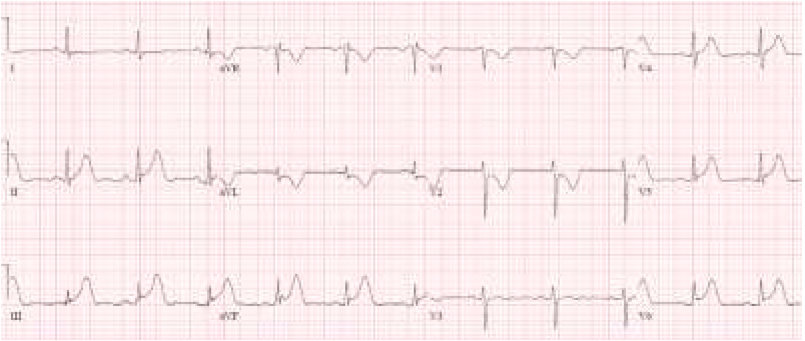

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What is the most likely diagnosis?

Anterolateral STEMI	4%
Inferolateral STEMI	77%
Myocarditis	4%

Pericarditis 3%

Posterior STEMI 13%

The ECG demonstrates ST elevation in the inferior (II, III, and aVF) and lateral leads (V5-6) confirming a diagnosis of an **inferolateral STEMI**. ST depression in V1-2 is suggestive of an associated posterior infarction. This constellation of ECG abnormalities is typically produced by occlusion of the proximal circumflex artery.

Anterolateral STEMI would present with ST elevation in the anterior (V1-V4) and lateral leads.

Posterior STEMI presents with reciprocal changes in the anterior leads including horizontal ST depression and a dominant R wave in V2.

Both **myocarditis and pericarditis** present with global concave ST elevation. The troponin is usually normal in pericarditis and raised in myocarditis.

Discuss (1) Improve

Next question ›

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	**Coronary artery**
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending

	ECG changes	**Coronary artery**
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: - horizontal ST depression - tall, broad R waves - upright T waves - dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

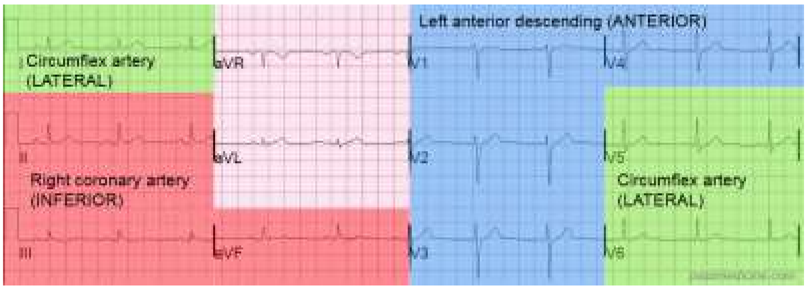
Circumflex artery (LATERAL)
I
aVR
V1
Left anterior descending (ANTERIOR)
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question >

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

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Extended textbook

Links

Postgraduate Medical Journal 👍 24 👎 40

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 14 👎 23

Classic circumflex infarction ECG

Suggest link

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Score: **8.3%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗
10	✗
11	✗
12	✗

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← Question 13 of 328 →

A 66-year-old lady attended a cardiology clinic for follow up after a recent hospital admission with pulmonary oedema. She continued to experience significant shortness of breath at rest with exercise tolerance that was limited to a few steps around the house. She had a previous medical history of hypertension, diabetes and myocardial infarction 5 years previously treated with percutaneous coronary intervention. Her medications comprised of bisoprolol 10mg, ramipril 10mg, furosemide 80mg twice daily, spironolactone 25mg, simvastatin 40mg, metformin 1g twice daily and aspirin 75mg. On examination she appeared breathless, the jugular venous pressure was elevated at 7cm, there were bibasal fine crepitations and moderate pitting oedema to her knees. Her heart rate was 68 beats per minute and blood pressure was 95/65mmHg.

Investigations:

Haemoglobin	115 g/L
White cell count	5.6 x10^9/L
Platelet Count	268 x10^9/L
Serum sodium	132mmol/L
Serum potassium	4.3mmol/L
Serum urea	6.7mmol/L
Serum creatinine	68micromol/L

Electrocardiogram: normal sinus rhythm. Rate 65 beats per minute. QRS duration 155ms. No acute ST changes.

Echocardiography: left ventricular ejection fraction of 30%. No significant valvular abnormalities.

Coronary angiography: Patent left anterior descending coronary artery stent. Minor diffuse coronary artery disease.

What is the most appropriate next management step?

○ Increase dose of furosemide ×

● Cardiac resynchronisation therapy ×

○ Coronary artery bypass graft ×

○ Start amlodipine ×

○ Home oxygen ×

Submit answer

Reference ranges ﹀

Score: **8.3%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
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10	✗
11	✗
12	✗
13	-

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Electrocardiogram: normal sinus rhythm. Rate 65 beats per minute. QRS duration 155ms. No acute ST changes.

Echocardiography: left ventricular ejection fraction of 30%. No significant valvular abnormalities.

Coronary angiography: Patent left anterior descending coronary artery stent. Minor diffuse coronary artery disease.

What is the most appropriate next management step?

Increase dose of furosemide 8%

Cardiac resynchronisation therapy	87%
Coronary artery bypass graft	2%
Start amlodipine	1%
Home oxygen	1%

This lady has ischaemic heart disease with New York Heart Association (NYHA) class 4 heart failure and is on maximal medical therapy. Her ECG shows a wide QRS complex which suggests ventricular dyssynchrony (i.e. The left and right ventricle are not beating at the same time). Resynchronisation of the ventricles with cardiac resynchronisation therapy with a paced device (CRT-P) involves simultaneous stimulation of both ventricles to reduce dyssynchrony and therefore increase cardiac output.

Nice supports the use of CRT-P in patients with:

- NYHA class three or four symptoms
- They are in normal sinus rhythm with either
- → QRS duration of 150 ms or longer estimated by standard electrocardiogram (ECG)
- → QRS duration of 120-149 ms estimated by ECG and mechanical dyssynchrony that is confirmed by echocardiography.
- They have a left ventricular ejection fraction of 35% or less.
- They are receiving optimal pharmacological therapy.

In this selected group of patients, it has been shown to improve symptoms by a reduction of about one NYHA classification, reduce hospitalisations and reduce mortality. These devices can be fitted with an implantable cardiac defibrillator (i.e. A CRT-D where the D stands for defibrillator as opposed to a CRT-P where P stands for paced and thus this device lacks the defibrillator component) for primary or secondary prophylaxis against ventricular dysrhythmias.

Increasing her dose of furosemide is unlikely to significantly improve her symptoms and is, therefore, an incorrect answer. Coronary artery bypass grafting has greater benefit than percutaneous coronary intervention at treating multivessel disease, however, this lady has disease of the left anterior descending artery which has been previously treated and only minor disease in her other coronary vessels; therefore this is an incorrect answer. She is hypotensive and therefore starting amlodipine is inappropriate. Home oxygen can be used for the palliation of shortness of breath in heart failure and is not appropriate in this case.

👍 👎 Discuss (13) Improve

Next question >

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy

- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training

- improves symptoms but not hospitalisation/mortality

AI + Q 123

Next question >

B I A

Textbooks

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Links

NICE 👍 14 👎 4

Cardiac resynchronisation therapy for the treatment of heart failure

NICE 👍 5 👎 1

2018 Chronic heart failure guidelines

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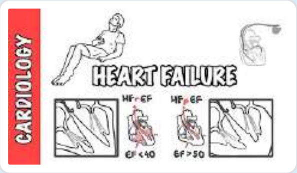
Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

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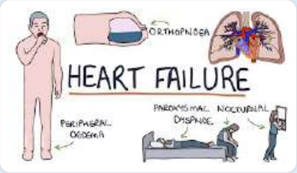
ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

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Score: **15.4%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
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9	✗
10	✗
11	✗
12	✗
13	✓

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Question 14 of 328

A 55-year-old with previous rheumatic heart disease aged 32 presents with an 18-month history of exertional dyspnoea. An initial echo demonstrated significant raised pulmonary arterial pressures of 77 mmHg, she undergoes a left and right heart catheter with results as follows:

		Oxygen saturations
Right atrium	8 mmHg	71%
Right ventricle	39/8 mmHg	71%
Pulmonary artery	45/12 mmHg	71%
Capillary wedge	20 mmHg	93%
Left ventricle	165/11 mmHg	93%
Aorta	90/58 mmHg	

What is the most likely diagnosis?

○ Aortic stenosis

○ Mitral stenosis

○ Aortic stenosis and mitral stenosis

○ Aortic stenosis and pulmonary hypertension

○ Aortic stenosis, mitral stenosis, pulmonary hypertension

Submit answer

Reference ranges

Score: **15.4%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗
10	✗
11	✗
12	✗
13	✓
14	-

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		Oxygen saturations
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Right ventricle	39/8 mmHg	71%
Pulmonary artery	45/12 mmHg	71%
Capillary wedge	20 mmHg	93%
Left ventricle	165/11 mmHg	93%
Aorta	90/58 mmHg	

What is the most likely diagnosis?

- Aortic stenosis 13%
- Mitral stenosis 7%
- Aortic stenosis and mitral stenosis 6%
- Aortic stenosis and pulmonary hypertension 47%
- Aortic stenosis, mitral stenosis, pulmonary hypertension 28%

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation and abnormalities in gradients across valves. There are no 'step-ups' in oxygen saturations, demonstrating no shunts. However, you will note that the pulmonary arterial pressure is greater than the normal one-fifth of systolic measurements; hence pulmonary hypertension is present. In addition, there is a greater than 25mmHg gradient across the aorta valve, demonstrating moderate aortic stenosis. Lastly, the capillary wedge pressure is equivalent to the left atrial pressure, which should also be the same as the left ventricular diastolic pressure. A normal mitral valve expects less than 5mmHg pressure difference. Using these inferences, the mitral valve gradient is calculated by the capillary wedge pressure of 20mmHg (same as the left atrial pressure) minus the diastolic left ventricular pressure of 11mmHg: the 9mmHg difference thus also demonstrates mitral stenosis.

👍 👎 Discuss (9) Improve

Next question >

Cardiac catheterisation and oxygen saturation levels ★

Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usually produce the answer.

Let's start with the basics:

- deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC). It has an oxygen saturation level of around **70%**. The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70%
- the lungs oxygenate the blood to a level of around **98-100%**. The left atrium (LA), left ventricle (LV) and aorta should all, therefore, have oxygen saturation levels of 98-100%

The table below shows the **oxygen saturations** that would be expected in different scenarios:

Diagnosis & notes	**RA**	**RV**	**PA**	**LA**	**LV**	**Aorta**
Normal	70%	70%	70%	100%	100%	100%
Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the	85%	85%	85%	100%	100%	100%

Diagnosis & notes	RA	RV	PA	LA	LV	Aorta
deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards						
Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated	70%	85%	85%	100%	100%	100%
Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PA having intermediate oxygenation levels	70%	70%	85%	100%	100%	100%
VSD with Eisenmenger's	70%	70%	70%	100%	85%	85%
PDA with Eisenmenger's	70%	70%	70%	100%	100%	85%
ASD with Eisenmenger's	70%	70%	70%	85%	85%	85%

+ Q 123

Next question ›

B *I* A

Textbooks

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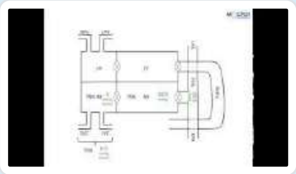
Media



Interpreting Cardiac Catheterisation Data

MRCPCH Revision - YouTube 👍 25 👎 5

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Score: **14.3%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗
10	✗
11	✗

12 ✗

13 ✓

14 ✗

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Question 15 of 328

A 72-year-old female presents with left chest pain associated with sweating in her hands and nausea. Her past medical history includes T2 diabetes mellitus, hypertension and hypercholesterolaemia, with her regular medications including metformin 850mg BD, gliclazide 40mg OD, ramipril 10mg, amlodipine 5mg and atorvastatin 40mg ON. On examination, her heart sounds are normal with a pansystolic murmur. Her ECG demonstrates ST depression in V2 to V5. Her BMs have ranged between 6 and 14 mmol/L since her admission 4 hours ago. How should you manage her diabetic medications?

- ○ No changes to medication
- ○ Increase metformin to 850mg TDS
- ○ Increase gliclazide to 40mg BD
- ○ Actrapid PRN 4 units when BM >11mmol/L
- ○ Stop all medications, start insulin sliding scale

Submit answer

Reference ranges

Score: **14.3%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

6	✗
7	✗
8	✗
9	✗
10	✗
11	✗
12	✗
13	✓
14	✗
15	-

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No changes to medication	15%
Increase metformin to 850mg TDS	2%
Increase gliclazide to 40mg BD	2%
Actrapid PRN 4 units when BM >11mmol/L	10%
Stop all medications, start insulin sliding scale	71%

Patients with acute coronary syndrome demonstrate significantly lower mortality rates at one year when treated with tight glycaemic control than without (19% vs 26%). Metformin has the potential to induce lactic acidosis in the context of any kind of ischaemia. An insulin based regime is recommended by the European Society of Cardiology[1], on the basis of results from DIGAMI trial[2]. PRN actrapid should never be prescribed routinely.

1. Wijns W, Kolh P, Danchin N et al. The task force on myocardial revascularisation of the European society of cardiology (ESC) and the European association for cardio-thoracic surgery (EACTS). European heart journal 2010; 31: 2501-55

2. Malmberg K. Prospective randomised study of intensive insulin treatment on long term survival after acute myocardial infarction in patients with diabetes mellitus. DIGAMI (Diabetes Mellitus, Insulin Glucose Infusion in Acute Myocardial Infarction) Study Group. BMJ.

1997;314(7093):1512

👍 | 👎 | Discuss (8) | Improve

Next question >

Myocardial infarction: STEMI management ★

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given
- **aspirin**
- **P2Y12-receptor antagonist**. Clopidogrel was the first P2Y12-receptor antagonist to be widely used but now ticagrelor is often favoured as studies have shown improved outcomes compared to clopidogrel, but at the expense of slightly higher rates of bleeding. This approached is supported in SIGN's 2016 guidelines. They also recommend that prasugrel (another P2Y12-receptor antagonist) could be considered if the patient is going to have a percutaneous coronary intervention
- **unfractionated heparin** is usually given for patients who're are going to have a PCI. Alternatives include low-molecular weight heparin

NICE suggest the following in terms of oxygen therapy:
- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is available.

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres. Thrombolysis should be performed in patients without access to primary

PCI

With regards to thrombolysis:
- tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase
- tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation
- if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis
- for patients successfully treated with thrombolysis PCI has been shown to be beneficial. The optimal timing of this is still under investigation

Glycaemic control in patients with diabetes mellitus
- in 2011 NICE issued guidance on the management of hyperglycaemia in acute coronary syndromes
- it recommends using a dose-adjusted insulin infusion with regular monitoring of blood glucose levels to glucose below 11.0 mmol/l
- intensive insulin therapy (an intravenous infusion of insulin and glucose with or without potassium, sometimes referred to as 'DIGAMI') regimes are not recommended routinely

+ Q 123

Next question >

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Links

NICE 👍 6 👎 9

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

SIGN 👍 7 👎 8

2016 Acute coronary syndrome guidelines

NICE 👍 10 👎 7

2011 Managing hyperglycaemia in acute coronary syndromes

Suggest link

Report broken link

Score: **13.3%**

1	✗
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15 ✗

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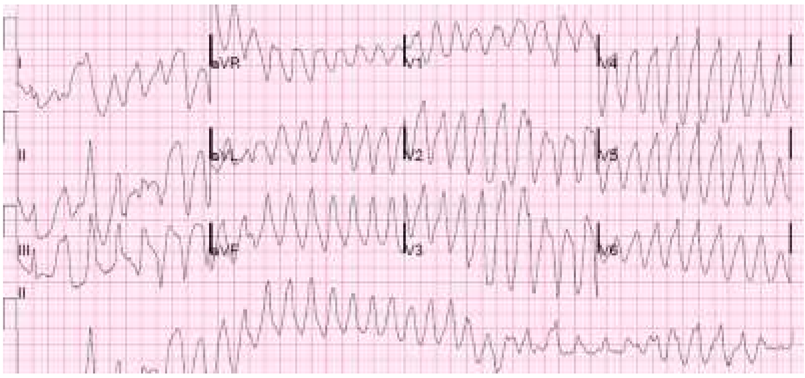
A 25-year-old man presents with a one-week history of feeling generally unwell, reporting myalgia, nausea and weakness.

His observations are:

- Respiratory rate: 24/min
- Blood oxygen saturation: 97% on room air
- Heart rate: 109bpm
- Blood pressure: 91/56mmHg
- Temperature: 37.7°C
- GCS 15/15

The patient is given intravenous anti-emetics and a bolus of crystalloid fluid. Whilst the fluid is running, the patient begins to complain of palpitations, chest pain and shortness of breath. An ECG is performed and is shown below.



© Image used on license from Dr Smith, University of Minnesota

What electrolyte abnormality is most likely to have precipitated this rhythm?

○ Hypercalcaemia ×

○ Hyperkalaemia ×

○ Hypermagnesaemia ×

○ Hypokalaemia ×

○ Hyponatraemia ×

Submit answer

Reference ranges

Score: **13.3%**

1	✗
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15	✗
16	-

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Question 16 of 328

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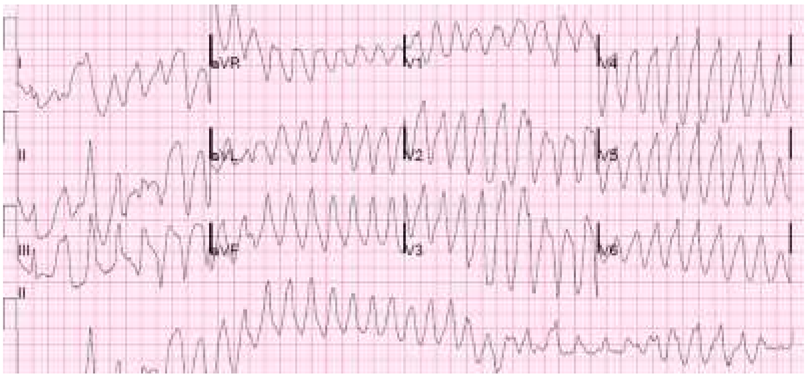

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What electrolyte abnormality is most likely to have precipitated this rhythm?

Hypercalcaemia	4%
Hyperkalaemia	15%
Hypermagnesaemia	4%
Hypokalaemia	75%

Hyponatraemia 1%

The ECG shows polymorphic ventricular tachycardia. This is shown by the broad complexes, and the QRS complexes varying in amplitude, axis and duration. The variation in QRS complexes is due to having multiple ventricular foci.

Hypokalaemia is the correct answer. Symptoms of myalgia and weakness are in keeping with hypokalaemia. Hypokalaemia can cause prolonged QT which can precipitate polymorphic ventricular tachycardia, as seen in the ECG. Hypokalaemia is the only electrolyte abnormality listed above that can precipitate prolonged QT.

Hypercalcaemia is the incorrect answer. Hypocalcaemia can precipitate prolonged QT.

Hyperkalaemia is the incorrect answer. As above, hypokalaemia can precipitate prolonged QT.

Hypermagnesaemia is the incorrect answer. Hypomagnesaemia can precipitate prolonged QT.

Hyponatraemia is the incorrect answer. Sodium derangement is not associated with prolonged QT and polymorphic ventricular tachycardia.

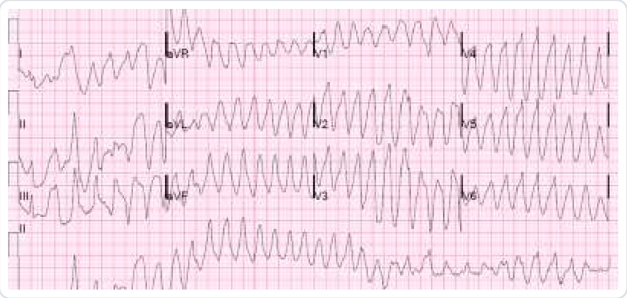
👍 👎 Discuss Improve

Next question >

Ventricular tachycardia ★

Ventricular tachycardia (VT) is broad-complex tachycardia originating from a ventricular ectopic focus. It has the potential to precipitate ventricular fibrillation and hence requires urgent treatment.

There are two main types of VT:
- monomorphic VT: most commonly caused by myocardial infarction
- polymorphic VT: A subtype of polymorphic VT is torsades de pointes which is precipitated by prolongation of the QT interval. The causes of a long QT interval are listed below



Causes of a prolonged QT interval

Congenital	Drugs	Other
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, fluoxetine • chloroquine • terfenadine • erythromycin	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Management

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy

- amiodarone: ideally administered through a central line
- lidocaine: use with caution in severe left ventricular impairment
- procainamide

Verapamil should NOT be used in VT

If drug therapy fails

- electrophysiological study (EPS)

- implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

123

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Links

Resuscitation Council UK 👍 10 👎 2

2021 Tachycardia algorithm

Suggest link Report broken link

Media

Ventricular tachycardia

Osmosis - YouTube 👍 2 👎 0

Ventricular Tachycardia (V-tach)
Khan Academy

Ventricular tachycardia

Khan Academy - YouTube 👍 1 👎 0

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Score: **12.5%**

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16	✗

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Question 17 of 328

A 25-year-old woman has recently been diagnosed with Marfan's syndrome. She has no other known diagnosis. She undergoes an echocardiogram which demonstrates severe aortic regurgitation, significant aortic root dilatation and a left ventricular ejection fraction of 70%. She is asymptomatic and has no family history of aortic dissection. What is the most appropriate management of her cardiac problem?

- ○ Follow-up in six months
- ○ ACE inhibitor
- ○ Exercise testing
- ○ Offer surgery if planning to become pregnant
- ○ Referral for aortic valve replacement

Submit answer

Reference ranges

Score: **12.5%**

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Question 17 of 328 ✓

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Follow-up in six months	26%
ACE inhibitor	12%
Exercise testing	3%
Offer surgery if planning to become pregnant	15%
Referral for aortic valve replacement ✓	45%

The correct answer is referral for aortic valve replacement. Surgery for aortic regurgitation is indicated if there is significant enlargement of the ascending aorta, severe regurgitation with symptoms or if severe with an ejection fraction of less than 50%. ACE inhibitors can be useful when there is hypertension, when surgery is contraindicated or when ventricular dysfunction persist following surgery. Exercise testing is more useful in aortic stenosis and follow-up would be acceptable if the aortic root was not dilated.

Source:
Vahanian A., Alfieri O., Andreotti F., et al. Guidelines on the management of valvular heart disease (version 2012). The Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery. European Heart Journal. 2012;33:24512496. doi: 10.1093/eurheartj/ehs109.

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Next question >

Aortic regurgitation ★

Aortic regurgitation (AR) is the leaking of the aortic valve of the heart that causes blood to flow in the reverse direction during ventricular diastole.

It can be caused either by disease of the aortic valve or by distortion or dilation of the aortic root and ascending aorta.

	Causes of AR due to valve disease	**Causes of AR due to aortic root disease**
Chronic presentation	• rheumatic fever: the most common cause in the developing world • calcific valve disease • connective tissue diseases e.g. rheumatoid arthritis/SLE • bicuspid aortic valve (affects both the valves and the aortic root)	• bicuspid aortic valve (affects both the valves and the aortic root) • spondylarthropathies (e.g. ankylosing spondylitis) • hypertension • syphilis • Marfan's, Ehler-Danlos syndrome
Acute presentation	• infective endocarditis	• aortic dissection

Features

- early diastolic murmur: intensity of the murmur is increased by the handgrip manoeuvre
- collapsing pulse
- wide pulse pressure
- Quincke's sign (nailbed pulsation)
- De Musset's sign (head bobbing)

- mid-diastolic Austin-Flint murmur in severe AR - due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams

Suspected AR should be investigated with echocardiography.

Management
- medical management of any associated heart failure
- surgery: aortic valve indications include
    - symptomatic patients with severe AR
    - asymptomatic patients with severe AR who have LV systolic dysfunction

+ Q 123

Next question >

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Media

www.thinklabs.com

Aortic regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 0 0

AORTIC VALVE DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **17.6%**

1	✗
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13	✓
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16	✗
17	✓

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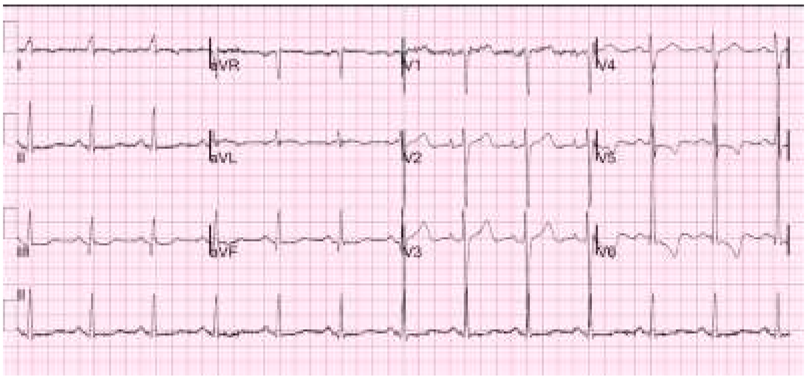
Question 18 of 328

A 52-year-old is brought from the renal outpatient clinic to the emergency department (ED) after appearing very short of breathing having walked up the stair. His breathlessness was associated with some mild, generalised chest discomfort, but the patient puts this down to a recent chest infection he has just recovered from.

When assessed in the ED the patient's breathlessness has improved and he denies any further chest discomfort. He reports he has been experiencing more frequent similar episodes, gradually increasing in severity over several months. He reports he recently had a productive cough with coryzal symptoms for the last 3-4 days but he feels it is improving. On questioning he confirms his symptoms of breathlessness are often worse during and post these chest infections.

He has a past medical history of chronic renal artery stenosis and resultant poorly controlled hypertension for which he is on multiple medications including an ACE inhibitor, beta-blocker and a diuretic.

An ECG is performed as seen below.



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What is the patient's most likely diagnosis?

○ Brugada syndrome ×

○ Hyperkalaemia ×

○ Left ventricular hypertrophy ×

○ Pericarditis ×

○ Wellen's syndrome ×

Submit answer

Reference ranges

Score: **17.6%**

1	✗
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18	-

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Question 18 of 328 ✓

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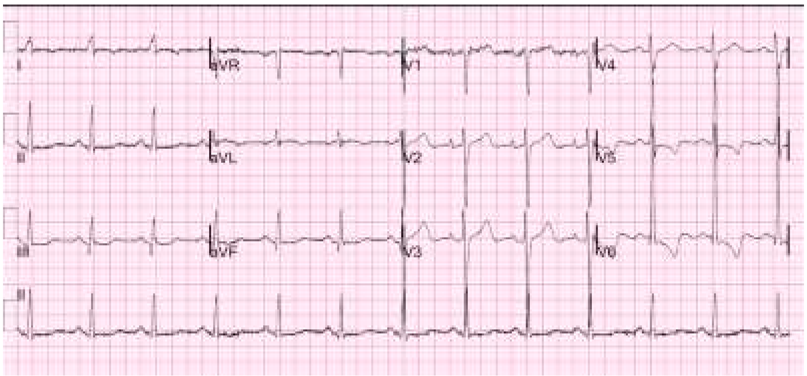

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What is the patient's most likely diagnosis?

- Brugada syndrome 8%
- Hyperkalaemia 3%
- Left ventricular hypertrophy 53%

Pericarditis	18%
Wellen's syndrome	19%

This patient has presented with symptoms and typical ECG changes in keeping with left ventricular (LV) hypertrophy. The thickening of the cardiac muscle is most likely a result of chronic uncontrolled hypertension, hence the prolonged, progressive history. LV hypertrophy results in signs and symptoms of LV failure including breathlessness, angina and reduced exercise tolerance. The patient's ECG demonstrates left ventricular hypertrophy with ST elevation in V2-3, without reciprocal lead changes and the combination of the S wave in V1 with the R wave in V5 or V6 clearly exceeding 40 mm.

Brugada syndrome is a genetic cardiac disorder resulting in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

There is no clear reason why this patient would be hyperkalaemia. Although he has renal impairment it is not indicated that he has end-stage renal failure or that he is on renal replacement therapy therefore hyperkalaemia would not be expected. Common ECG changes noted in hyperkalaemia included peaked T waves, P wave widening/flattening, PR prolongation progressing to the bradyarrhythmias and conductions blocks. No clear features of hyperkalaemia can be seen on this patient's ECG.

Pericarditis is inflammation of the tissue surrounding the heart and can be due to infections. Patients normally present with pleuritic, constant chest pain and fevers. Although this patient has a very recent chest infection, pericarditis normally develops a few weeks post the resolution of infection as it is an immune response and not a direct result of the infection itself. ECG findings associated with pericarditis include widespread concave ST elevation and PR depression which are not present on this patient's ECG.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending (LAD) artery and therefore is normally seen in patients presenting with ischaemic like symptoms. You would expect the patient's chest pain to be continuous and to be more severe if he was experiencing stenosis of the LAD.

Discuss (7) | Improve

Next question >

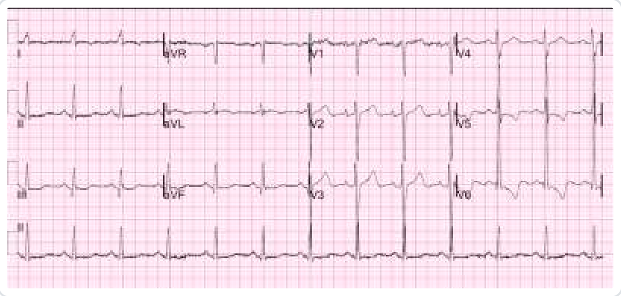
ECG: atrial and ventricular hypertrophy ★

Left ventricular hypertrophy
- sum of S wave in V1 and R wave in V5 or V6 exceeds 40 mm

Right ventricular hypertrophy

Left atrial enlargement
- bifid P wave in lead II with a duration > 120 ms
- in V1 the P wave has a negative terminal portion

Right atrial enlargement
- tall P waves in both II and V1 which exceed 0.25 mV



+ Q 123

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Score: **22.2%**

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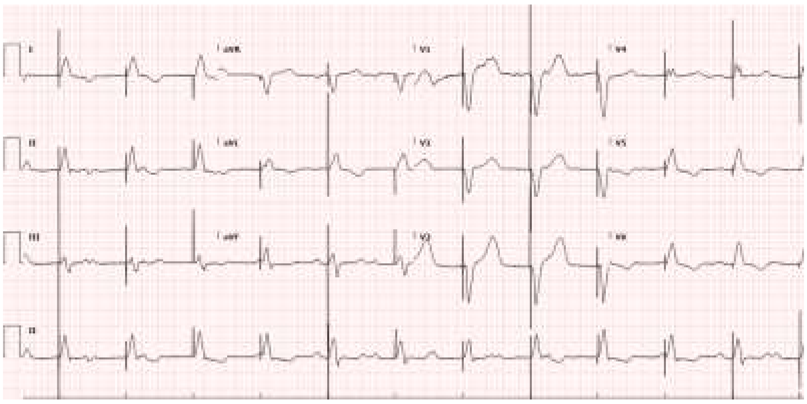
A 62-year-old man is recovering from an aortic valve replacement via median sternotomy in the high-dependency unit. You are called to see him overnight as he is experiencing chest pain, worse on breathing. An ECG is performed and displayed below. He had temporary trans-venous pacing wires inserted during the procedure.



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What does the ECG show?

- ○ AAI pacing ×
- ○ Complete heart block with loss of capture ×
- ○ DDD pacing ×
- ○ Myocardial infarction ×
- ○ VVI pacing ×

Submit answer

Reference ranges

Score: **22.2%**

1	✗
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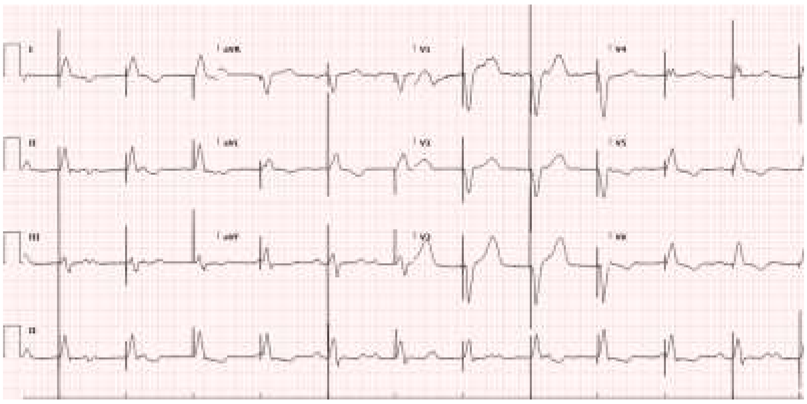

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What does the ECG show?

AAI pacing	10%
Complete heart block with loss of capture	15%
DDD pacing	15%
Myocardial infarction	5%
VVI pacing	54%

Although atrial pacing wires are commonly inserted following cardiac surgery, it is particularly important to insert ventricular wires following aortic valve surgery due to the significantly higher incidence of complete heart block postoperatively. The ECG shows pacing spikes immediately preceding QRS complexes, indicating ventricular **(VVI)** pacing with good capture. This patient has a degree of heart block underlying his paced rhythm, hence the discordance between the p waves and the QRS complexes, and the reason why the patient is paced.

AAI pacing would show pacing spikes prior to p waves, and in instances of heart block as shown here there would be abnormal or potentially absent conduction to the ventricles. As the pacing spikes are associated with the QRS complex here, AAI cannot be the mode in use.

Although this patient may have **complete heart block**, there is good capture as shown by a 1:1 ratio between pacing spikes and subsequent QRS complexes. Therefore loss of capture is an incorrect answer.

DDD pacing refers to the pacing of both the atria and ventricles. Although this can be useful post-operatively to increase cardiac output, it is not being used here. DDD pacing would either show pacing of the atria (pacing spikes before p waves) or tracking of the patient's intrinsic atrial rate (ventricular pacing spikes that follow p waves and immediately precede QRS complexes).

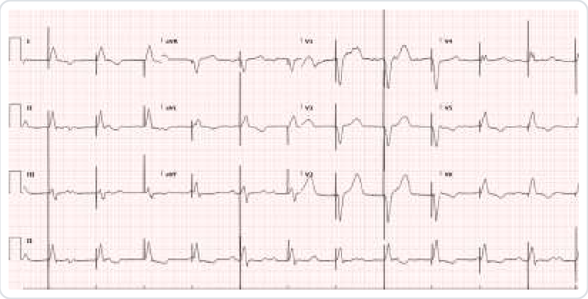
Pain is very common following a sternotomy and is classically sharp and pleuritic. It can be difficult to interpret ischaemia on a paced ECG however the Sgarbossa criteria is a useful tool for this. As this patient does not fit this criterion, and the pain is classical for sternotomy pain rather than cardiac, **myocardial infarction** is unlikely.

👍 👎 Discuss (2) Improve

Next question >

Pacemakers: temporary ★

Indications for a temporary pacemaker
- symptomatic/haemodynamically unstable bradycardia, not responding to atropine
- post-ANTERIOR MI: type 2 or complete heart block
    - post-INFERIOR MI complete heart block is common and can be managed conservatively if asymptomatic and haemodynamically stable
- trifascicular block prior to surgery



123

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Links

Resuscitation Council UK 👍 15 👎 2

2021 Bradycardia algorithm

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Score: **21.1%**

1 ✗

2 ✗

3	✗
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19	✗

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Question 20 of 328

A 68 year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is 2 days post right curative hemicolectomy for bowel malignancy.

He has developed chest pain and shortness of breath. The surgical team have requested an urgent medical review.

On examination the patient appears distressed and is complaining of chest pain and breathlessness. Capillary refill time is 5 seconds centrally. The pulse rate is 200bpm and the blood pressure is 87/45mmHg. Oxygen saturations are 92% on fiO2 0.4 via Venturi mask.

There are crackles to the midzones bilaterally on chest auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform.

Blood results from the morning reveal:

Hb	129 g/l
Platelets	$564 * 10^9$/l
WBC	$15.8 * 10^9$/l
Na^+	128 mmol/l
K^+	3.3 mmol/l
Urea	12.9 mmol/l
Creatinine	101 µmol/l
Bilirubin	27 µmol/l
ALP	125 u/l
ALT	34 u/l
Albumin	36 g/l

What is the most appropriate initial management?

○ Adenosine 6mg IV ×

○ Amiodarone 300mg IV ×

○ Magnesium sulphate 2g IV ×

○ Synchronised DC shock ×

○ Metoprolol 5mg IV ×

Submit answer

Reference ranges ∨

Score: **21.1%**

1	✗
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Question 20 of 328 ✗

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Creatinine	101 µmol/l
Bilirubin	27 µmol/l
ALP	125 u/l
ALT	34 u/l
Albumin	36 g/l

What is the most appropriate initial management?

Adenosine 6mg IV 1%

Amiodarone 300mg IV	6%
Magnesium sulphate 2g IV	5%
Synchronised DC shock	87%
Metoprolol 5mg IV	0%

In the context of a tachyarrhythmia, a systolic BP < 90 mmHg → DC cardioversion

Important for me Less important

This question outlines a case of ventricular tachycardia with adverse features including shock and heart failure.

According to the Resuscitation Council guidelines, if a patient with a tachyarrhythmia is unstable (i.e. has adverse features likely to be caused or made worse by the tachycardia - shock, syncope, heart failure or myocardial ischaemia) synchronised cardioversion is the treatment of choice.

If cardioversion fails to restore sinus rhythm, and the patient remains unstable, the next step is to give amiodarone 300 mg IV over 10-20 min and re-attempt electrical cardioversion. The loading dose of amiodarone may be followed by an infusion of 900 mg over 24 h.

If the patient is conscious, cardioversion should be carried out under sedation or general anaesthesia in the presence of an anaesthetist.

Source:
https://www.resus.org.uk/resuscitation-guidelines/peri-arrest-arrhythmias/#tachycardia

👍 👎 Discuss (5) Improve

Next question >

Peri-arrest rhythms: tachycardia ★

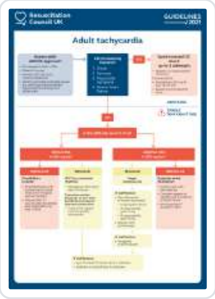
The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given. Up to 3 shocks can be given; after this expert help should be sought.

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment guidelines can be found at the Resuscitation Council website, below is a very limited summary:



Broad-complex tachycardia

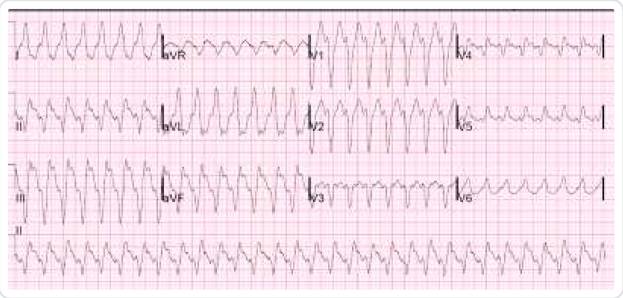
Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion

Irregular

- seek expert help. Possibilities include:

  - atrial fibrillation with bundle branch block - the most likely cause in a stable patient
  - atrial fibrillation with ventricular pre-excitation
  - torsade de pointes


aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

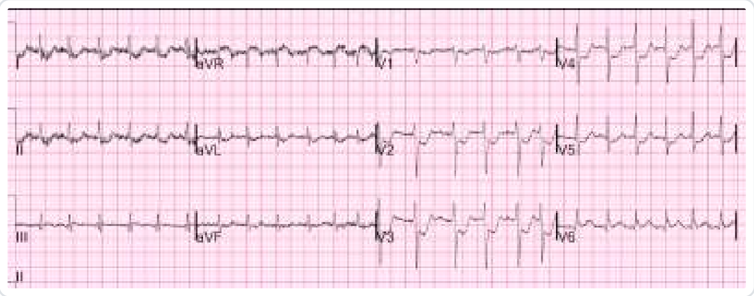
Narrow-complex tachycardia

Regular

- vagal manoeuvres followed by IV adenosine
- if the above is unsuccessful consider a diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control: beta-blockers are usually first-line unless there is a contraindication


aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

123

Next question >

Textbooks

High-yield textbook

Extended textbook

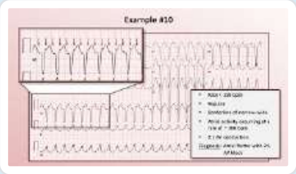
Links

Resuscitation Council UK 👍 30 👎 9

2021 Tachycardia algorithm

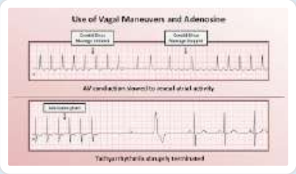
Suggest link Report broken link

Media



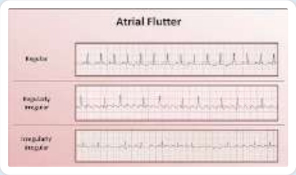
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 5 👎 0


Use of Vagal Maneuvers and Adenosine

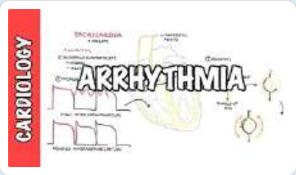
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 4 👎 0


Atrial Flutter

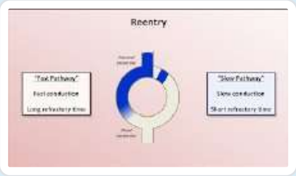
Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 1


CARDIOLOGY
TACHYCARDIA
ARRHYTHMIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 1


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 2 👎 1

Report broken media

Score: **20%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✓
14 ✗

15	✗
16	✗
17	✓
18	✓
19	✗
20	✗

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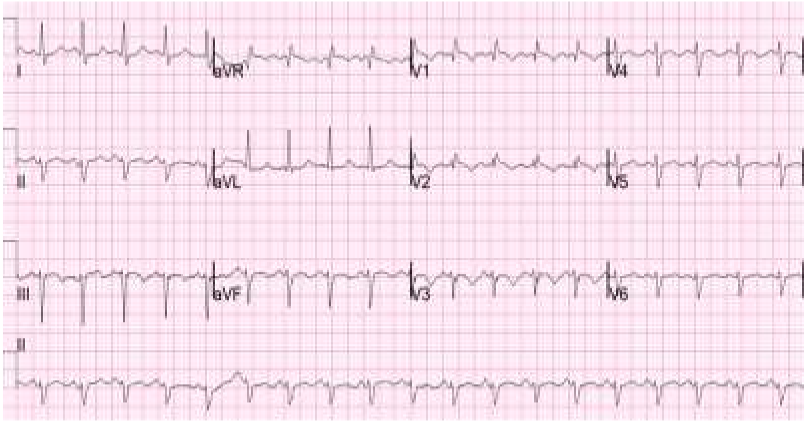
You are called to review a 68-year-old who is complaining of chest pain on the ward. She was admitted 6 days ago after having a stroke which has left her with a mild degree of right-sided hemiparesis. She has some difficultly articulating the nature of the pain but describes it as central.

Her past medical history includes hypertension and hypothyroidism. She has not had a stroke or transient ischaemic attack previously.

On examination, the heart rate is 102/min, blood pressure 122/78 mmHg, respiratory rate 22/min and oxygen saturations 94% on room air.

The nurse recorded an ECG:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

○ Subarachnoid haemorrhage

○ Inferior myocardial infarction

○ Hypokalaemia

○ Posterior myocardial infarction

○ Pulmonary embolism

Submit answer

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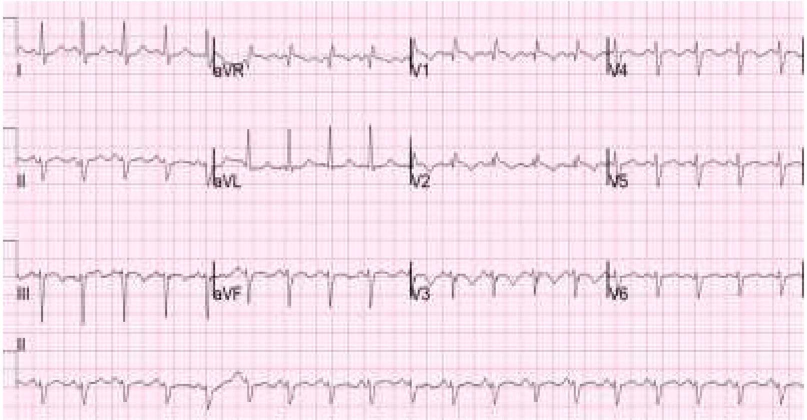


© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Subarachnoid haemorrhage	7%
Inferior myocardial infarction	6%
Hypokalaemia	10%
Posterior myocardial infarction	21%
Pulmonary embolism	56%

S1Q3T3 is a classic but uncommon ECG finding in PE

Important for me Less important

This woman is at risk of a pulmonary embolism given her relative immobility following the stroke.

The tachycardia and tachypnoea are also signs associated with pulmonary embolism.

The ECG shows some of the features that may be associated with pulmonary embolism (sinus tachycardia, RBBB, right ventricular strain). Other features such as the left axis deviation are atypical.

👍 👎 Discuss (8) Improve

Next question >

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:

- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

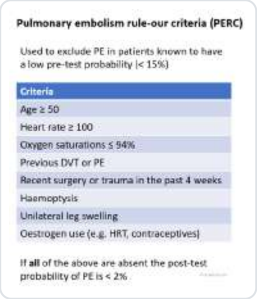
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
  - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3

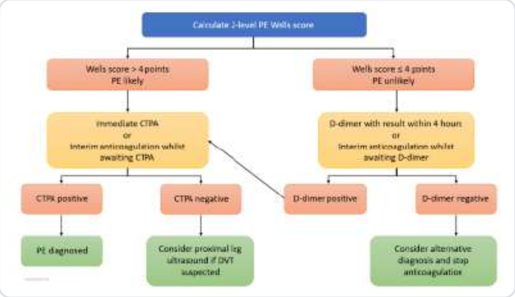
Clinical feature	**Points**
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

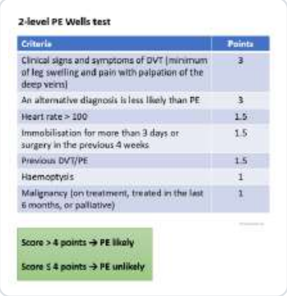
Clinical probability simplified scores
- PE likely - more than 4 points
- PE unlikely - 4 points or less

If a PE is 'likely' (more than 4 points)
- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
    - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)
- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed

  - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points
PE likely
Wells score ≤ 4 points
PE unlikely
Immediate CTPA
or
Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours
or
Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

Some other points

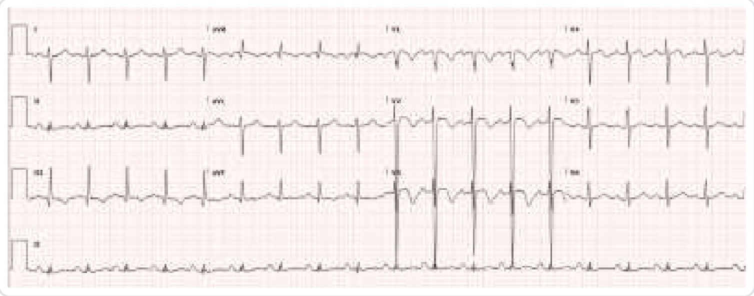
D-dimers

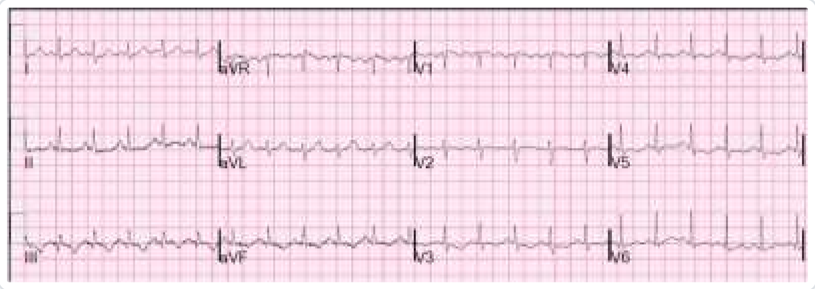
- sensitivity = 95-98%, but poor specificity

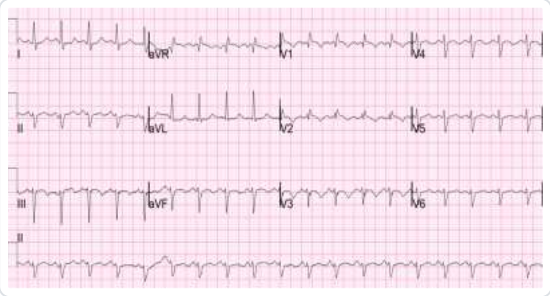
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen







Chest x-ray

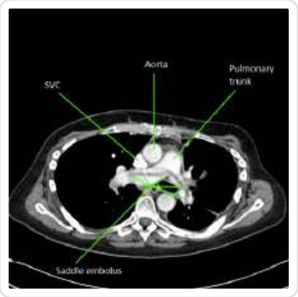
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

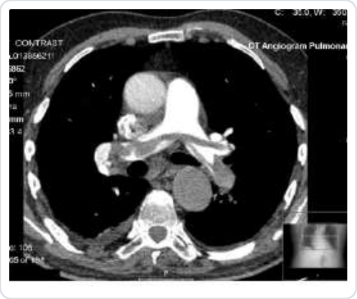
V/Q scan

- sensitivity of around 75% and specificity of 97%

- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA
- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus



1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 13 👎 10

The ECG in Pulmonary Embolism

MDcalc 👍 15 👎 8

Wells' Criteria for Pulmonary Embolism

MDcalc 👍 17 👎 8

PERC Rule for Pulmonary Embolism

NICE 👍 20 👎 9

2020 Venous thromboembolism guidelines

Suggest link

Report broken link

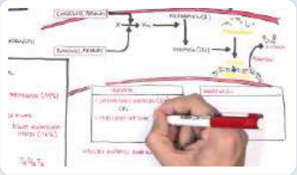
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

Report broken media

Score: **19%**

1	✗
2	✗
3	✗
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5	✓
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14	✗
15	✗
16	✗
17	✓
18	✓
19	✗
20	✗
21	✗

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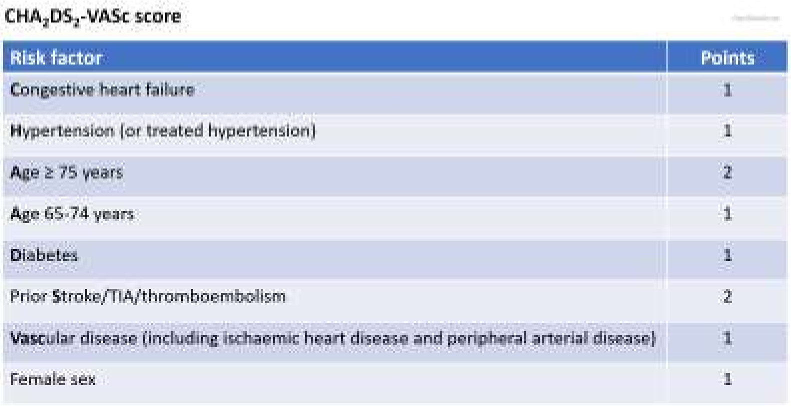
A 76-year-old woman presents to the acute medical assessment unit with a two-month history of palpitations. She reports regularly feeling her heart racing, occasionally associated with pre-syncope. She denies dyspnoea, syncope and chest pain.

She has a medical history significant for asthma, for which she takes regular inhaled beclometasone dipropionate and formoterol, and montelukast, with as required salbutamol. There is no other medical history.

Her observations are:

- Respiratory rate: 20/min
- Blood oxygen saturation: 98% on room air
- Heart rate: 116bpm
- Blood pressure: 135/68mmHg
- GCS: 15/15
- Temperature: 37.4ºC

An ECG is performed which shows atrial fibrillation. Blood tests are unremarkable. A transthoracic echocardiogram shows no evidence of valvular heart disease and no evidence of congestive cardiac failure.



CHA_2DS_2-VASc score

Risk factor	Points
Congestive heart failure	1
Hypertension (or treated hypertension)	1
Age ≥ 75 years	2
Age 65-74 years	1
Diabetes	1
Prior Stroke/TIA/thromboembolism	2
Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
Female sex	1

What is the most appropriate management?

○ Apixaban + bisoprolol

○ Apixaban + digoxin ×

○ Apixaban + diltiazem ×

○ Warfarin + bisoprolol ×

○ Warfarin + diltiazem ×

Submit answer

Reference ranges ⌄

Score: **19%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
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12	✗
13	✓
14	✗
15	✗
16	✗
17	✓
18	✓
19	✗

20	✗
21	✗
22	-

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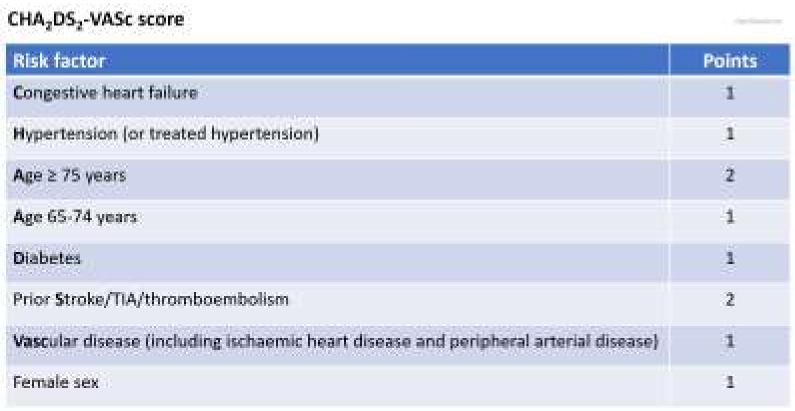

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Age ≥ 75 years	2
Age 65-74 years	1
Diabetes	1
Prior Stroke/TIA/thromboembolism	2
Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
Female sex	1

What is the most appropriate management?

Apixaban + bisoprolol 30%

Apixaban + digoxin	13%
Apixaban + diltiazem	55%
Warfarin + bisoprolol	2%
Warfarin + diltiazem	2%

Apixaban + diltiazem is the correct answer. This patient has atrial fibrillation with no valvular abnormalities. Her CHA_2DS_2-VASc score is 3 and therefore qualifies for anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban. If this patient had mitral stenosis, weighed over 120kg, or had a contraindication to a DOAC, warfarin would be considered. This patient would be suitable for rate control rather than rhythm control - the two-month history suggests this is not acute, there is no clear reversible cause and there is no evidence of heart failure. This patient's asthma is a contra-indication for a beta-blocker, as beta-blockers can increase airway reactivity. Calcium channel blockers are second-line for rate control in atrial fibrillation. Therefore apixaban + diltiazem is the correct combination of medications.

Apixaban + bisoprolol is the incorrect answer. Beta-blockers are the first-line treatment for rate control in atrial fibrillation, but are relatively contraindicated in asthma - given this patient is on fourth-line treatment for her asthma, beta-blockers are best avoided in this scenario.

Apixaban + digoxin is the incorrect answer. Digoxin is the third-line treatment for rate control in atrial fibrillation. Therefore a calcium channel blocker such as diltiazem is more appropriate. Digoxin would be more appropriate if this patient had evidence of heart failure due to the negative inotropic effects of calcium channel blockers.

Warfarin + bisoprolol is the incorrect answer. DOACs, such as apixaban, are used first line when anticoagulation is indicated for atrial fibrillation. Warfarin is recommended if there is evidence of mitral stenosis - however, this patient's echocardiogram has no evidence of valvular disease. As above, diltiazem is more appropriate than bisoprolol due to this patient's asthma.

Warfarin + diltiazem is the incorrect answer. As above, a DOAC is preferable to warfarin in this situation. Diltiazem is the correct drug for rate control in this scenario.

👍 👎 Discuss (3) Improve

Next question ›

Atrial fibrillation: anticoagulation ★

NICE updated their guidelines on the management of atrial fibrillation (AF) in 2021.

Does the patient need anticoagulation?

The need for anticoagulation should be considered in patients with any history of AF, not just if they are in AF currently, i.e. the following groups should be assessed:

- symptomatic or asymptomatic paroxysmal, persistent or permanent atrial fibrillation
- atrial flutter
- a continuing risk of arrhythmia recurrence after cardioversion back to sinus rhythm or catheter ablation

NICE suggest using the **CHA_2DS_2-VASc** score to determine the most appropriate anticoagulation strategy. This scoring system superceded the $CHADS_2$ score.

	Risk factor	**Points**
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A_2	Age >= 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S_2	Prior Stroke, TIA or thromboembolism	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1

	Risk factor	Points
S	Sex (female)	1

The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender)
2 or more	Offer anticoagulation

Remember that if a CHA_2DS_2-VASc score suggests no need for anticoagulation it is important to ensure a transthoracic echocardiogram has been done to exclude valvular heart disease, which in combination with AF is an absolute indication for anticoagulation.

Assessing bleeding risk

Doctors have always thought carefully about the risk/benefit profile of starting someone on warfarin. A history of falls, old age, alcohol excess and a history of previous bleeding are common things that make us consider whether anticoagulation is in the best interests of the patient., but NICE warn us not to withhold anticoagulation solely on the grounds of age or risk falls. NICE now recommend we formalise this risk assessment using the ORBIT scoring system. Previously the HAS-BLED scoring system was recommended.

Variable	Points
Haemoglobin <130 g/L for males and < 120 g/L for females, or haemtocrit < 40% for males and < 36% for females	2
Age > 74 years	1

Variable	Points
Bleeding history (GI bleeding, intracranial bleeding or haemorrhagic stroke)	2
Renal impairment (GFR < 60 mL/min/1.73m^2)	1
Treatment with antiplatelet agents	1

There are no formal rules on how we act on the ORBIT score and individual patient factors should be taken into account. However, the following table acts as a guide:

ORBIT score	Risk group	Bleeds per 100 patient-years
0-2	Low	2.4
3	Medium	4.7
4-7	High	8.1

If treatment is indicated, which anticoagulant?

For many years warfarin was the anticoagulant of choice in atrial fibrillation. This changed following the development of **direct oral anticoagulants (DOACs)**. These have the advantage of not requiring regular blood tests to check the INR and now recommended as the first-line anticoagulant for patients with AF. If a patient was previously started on warfarin, NICE recommend discussing switching to a DOAC during a routine follow-up appointment.

The following DOACs are recommended by NICE for reducing stroke risk in AF:
- apixaban
- dabigatran
- edoxaban
- rivaroxaban

Warfarin is now used second-line, in patients where a DOAC is contraindicated or not tolerated.

Aspirin is not recommended for reducing stroke risk in patients with AF.

AI + Q 123

Next question >

Textbooks

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Extended textbook

Links

European Society of Cardiology 👍 13 👎 14

2020 Atrial fibrillation guidelines

NICE 👍 18 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

SPARCtool 👍 28 👎 8

Stroke Prevention in Atrial Fibrillation Risk Tool

NICE 👍 43 👎 9

2021 Atrial fibrillation guidelines

Suggest link

Report broken link

Score: **18.2%**

1	✗
2	✗
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17	✓
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21	✗
22	✗

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A 78-year-old female was referred to the clinic after several episodes of chest pain. The chest pain came on exertion and was relieved by sitting down. The pain was centrally located and described as crushing in character.

She has a past medical history of hypercholesterolaemia, systemic lupus erythematosus and hypertension.

A myocardial technetium (99mTc) MIBI scan demonstrates the following results:

At rest	Normal perfusion
Under stress	Lateral perfusion defect

What is the most likely explanation of these results?

- ○ Myocardial infarction of left circumflex artery territory
- ○ Myocardial infarction of left anterior descending coronary artery territory
- ○ 70% stenosis of left circumflex artery
- ○ 70% stenosis of left anterior descending coronary artery
- ○ Libman-Sacks endocarditis

Submit answer

Reference ranges

Score: **18.2%**

1	✗
2	✗
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Question 23 of 328 ✗

A 78-year-old female was referred to the clinic after several episodes of chest pain. The chest pain came on exertion and was relieved by sitting down. The pain was centrally located and described as crushing in character.

She has a past medical history of hypercholesterolaemia, systemic lupus erythematosus and hypertension.

A myocardial technetium (99mTc) MIBI scan demonstrates the following results:

At rest	Normal perfusion
Under stress	Lateral perfusion defect

What is the most likely explanation of these results?

Myocardial infarction of left circumflex artery territory	8%
Myocardial infarction of left anterior descending coronary artery territory	2%
70% stenosis of left circumflex artery	76%
70% stenosis of left anterior descending coronary artery	10%
Libman-Sacks endocarditis	4%

If a cardiac MIBI scan shows that the defect is present on stress, but not at rest, this is suggestive of a reversible defect (e.g. myocardial ischaemia secondary to coronary artery stenosis)

Important for me Less important

A myocardial perfusion (MIBI) scan is carried out to investigate the blood supply to the heart. By comparing the rest with stress images, any areas of ischaemia can classified as reversible or fixed.

If the defect is present on stress, but not at rest, then this is suggestive of a reversible defect (e.g. myocardial ischaemia secondary to coronary artery stenosis).

As demonstrated in this case, the defect is present only on stress, which is indicative of a reversible defect such as coronary artery stenosis.

The lateral part of the heart is usually supplied by the left circumflex artery, making this the best answer.

👍 👎 Discuss (4) Improve

Next question >

Cardiac imaging: non-invasive techniques excluding echocardiography ★

The ability to image the heart using non-invasive techniques such as MRI, CT and radionuclides has evolved rapidly over recent years.

Nuclear imaging

These techniques use radiotracers which are extracted by normal myocardium. Examples include:

- thallium
- technetium (99mTc) sestamibi: a coordination complex of the radioisotope technetium-99m with the ligand methoxyisobutyl isonitrile (MIBI), used in 'MIBI' or cardiac Single Photon Emission Computed Tomography (SPECT) scans
- fluorodeoxyglucose (FDG): used in Positron Emission Tomography (PET) scans

The primary role of SPECT is to assess myocardial perfusion and myocardial viability. Two sets of images are usually acquired. First the myocardium at rest followed by images of the myocardium during stress (either exercise or following adenosine / dipyridamole). By comparing the rest with stress images any areas of ischaemia can classified as reversible or fixed (e.g. Following a myocardial infarction). Cardiac PET is predominately a research tool at the current time

MUGA

- Multi Gated Acquisition Scan, also known as radionuclide angiography
- radionuclide (technetium-99m) is injected intravenously
- the patient is placed under a gamma camera
- may be performed as a stress test
- can accurately measure left ventricular ejection fraction. Typically used before and after cardiotoxic drugs are used

Cardiac Computed Tomography (CT)

Cardiac CT is useful for assessing suspected ischaemic heart disease, using two main methods:

- calcium score: there is known to be a correlation between the amount of atherosclerotic plaque calcium and the risk of future ischaemic events. Cardiac CT can quantify the amount of calcium producing a 'calcium score'
- contrast enhanced CT: allows visualisation of the coronary artery lumen

If these two techniques are combined cardiac CT has a very high negative predictive value for ischaemic heart disease.

Cardiac MRI

Cardiac MRI (commonly termed CMR) has become the gold standard for providing structural images of the heart. It is particularly useful when assessing congenital heart disease, determining right and left ventricular mass and differentiating forms of cardiomyopathy. Myocardial perfusion can also be assessed following the administration of gadolinium. Currently CMR provides limited data on the extent of coronary artery disease.

Please also see the British Heart Foundation link for an excellent summary.

AI + Q 123

Next question ›

Textbooks

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Links

NICE 👍 2 👎 4

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Royal College of Physicians 👍 3 👎 2

2012 Nuclear cardiology review

Suggest link Report broken link

Score: **17.4%**

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A 46-year-old man is referred to the acute medical assessment unit with a fever of 38.9 °C. He reports a three-week history of general malaise, reduced appetite, intermittent sweating, and occasional rigors. The patient has no medical history and has had no previous operations or procedures. He denies any drug allergies.

His observations are:

- Respiratory rate: 24/min
- Oxygen saturation: 95% on room air
- Heart rate: 105bpm
- BP: 101/62mmHg
- Temperature: 38.9 °C
- GCS: 15/15

On examination, the patient has a loud early diastolic murmur over the right second intercostal space. Splinter haemorrhages are noted in three of his fingernails.

What is the most appropriate antibiotic to start?

○ Amoxicillin

○ Benzylpenicillin

○ Flucloxacillin

○ Vancomycin + low-dose gentamicin

○ Vancomycin + low-dose gentamicin + rifampicin

Submit answer

Reference ranges

A 46-year-old man is referred to the acute medical assessment unit with a fever of 38.9 °C. He reports a three-week history of general malaise, reduced appetite, intermittent sweating, and occasional rigors. The patient has no medical history and has had no previous operations or procedures. He denies any drug allergies.

His observations are:

- Respiratory rate: 24/min
- Oxygen saturation: 95% on room air
- Heart rate: 105bpm
- BP: 101/62mmHg
- Temperature: 38.9 °C
- GCS: 15/15

On examination, the patient has a loud early diastolic murmur over the right second intercostal space. Splinter haemorrhages are noted in three of his fingernails.

What is the most appropriate antibiotic to start?

Amoxicillin	43%
Benzylpenicillin	11%
Flucloxacillin	16%
Vancomycin + low-dose gentamicin	24%
Vancomycin + low-dose gentamicin + rifampicin	6%

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

Important for me Less important

Fever + a new cardiac murmur raises suspicion of infective endocarditis. A loud early diastolic murmur over the right second intercostal space is suspicious for aortic regurgitation secondary to endocarditis. This will cause the patient's shortness of breath.

Amoxicillin is the correct answer. Given the patient has no medical or surgical history, he should be treated for native valve endocarditis by starting high-dose intravenous amoxicillin. Ideally, six sets of blood cultures should be sent, with as many as possible before the initiation of treatment (although this should not delay treatment if the patient is unstable).

Benzylpenicillin is the incorrect answer. Benzylpenicillin is the treatment for infective endocarditis caused by a fully sensitive Streptococcus organism.

Flucloxacillin is the incorrect answer. Flucloxacillin is used to treat infective endocarditis caused by a fully sensitive Staphylococcus (usually *Staphylococcus aureus*.)

Vancomycin + low-dose gentamicin is the incorrect answer. Vancomycin + low-dose gentamicin can be used for infective endocarditis in penicillin-allergic patients or patients with methicillin-resistant *Staphylococcus aureus* (MRSA) infective endocarditis.

Vancomycin + low-dose gentamicin + rifampicin is the incorrect answer. Vancomycin + low-dose gentamicin + rifampicin is the treatment for infective endocarditis on a prosthetic valve. This patient has no history of valve replacement.

👍 👎 Discuss (4) Improve

Next question >

Infective endocarditis: prognosis and management ★

Poor prognostic factors

- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism

- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin If prosthetic valve • vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA • vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA • vancomycin + rifampicin + low-dose gentamicin

Scenario	Suggested antibiotic therapy
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic • vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic • vancomycin + low-dose gentamicin

Indications for surgery
- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

AI + Q 123

Next question ›

Textbooks

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Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **16.7%**

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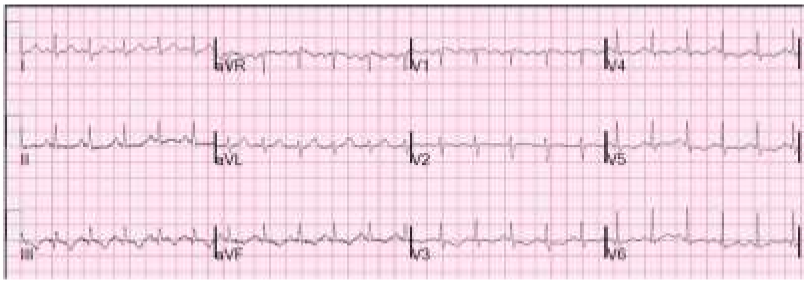
A 19-year-old student is brought to the Emergency Department by her friends. Around one hour ago she 'collapsed' whilst playing hockey. Her friends describe her complaining that she felt light-headed and then 'fainting' to the ground. She lost consciousness for a few seconds before returning to normal quite quickly. There is no past medical history of note other than the use of Microgynon 30 (a combined oral contraceptive pill). For the past 4-5 days the patient has experienced shortness-of-breath and a central chest pain which is worse when she coughs. On examination her pulse is 120/min, blood pressure 96/60 mmHg and chest auscultation reveals scattered wheezes. An ECG done on admission is shown below:



© Image used on license from Dr Smith, University of Minnesota

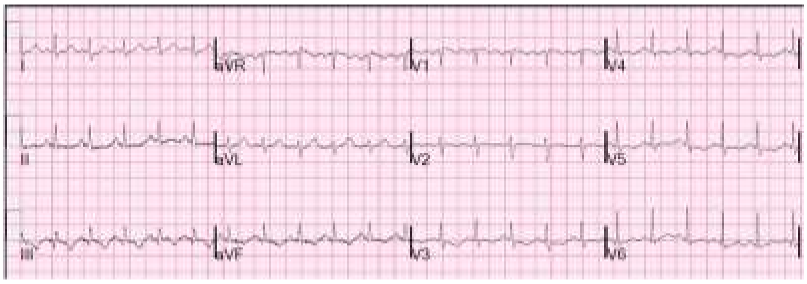
What is the most likely diagnosis?

- ○ Hypertrophic obstructive cardiomyopathy
- ○ Vasovagal attack
- ○ Acute coronary syndrome
- ○ Pulmonary embolism
- ○ Asthma attack

Submit answer

Reference ranges

A 19-year-old student is brought to the Emergency Department by her friends. Around one hour ago she 'collapsed' whilst playing hockey. Her friends describe her complaining that she felt light-headed and then 'fainting' to the ground. She lost consciousness for a few seconds before returning to normal quite quickly. There is no past medical history of note other than the use of Microgynon 30 (a combined oral contraceptive pill). For the past 4-5 days the patient has experienced shortness-of-breath and a central chest pain which is worse when she coughs. On examination her pulse is 120/min, blood pressure 96/60 mmHg and chest auscultation reveals scattered wheezes. An ECG done on admission is shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Hypertrophic obstructive cardiomyopathy	15%
Vasovagal attack	5%
Acute coronary syndrome	1%
Pulmonary embolism	78%
Asthma attack	2%

It is usual taught that pulmonary embolism (PE) presents with pleuritic chest pain, dyspnoea and haemoptysis. This combination of symptoms is however only found in less than 20% of cases. As PE is a potentially life-threatening condition it is important to be aware of the wide variety of symptoms and signs that may accompany cases.

A lot of patients who develop a PE have risk factors. There is one present in this case - combined oral contraceptive pill use. Tachycardia is also a common sign.

It would be unusual to develop an asthma attack with no previous history of asthma. Occasional wheezes are a relatively common finding in patients following a pulmonary embolism.

The ECG shows a sinus tachycardia and a partial S1Q3T3 - the S wave is not particularly convincing.

👍 👎 Discuss (5) Improve

Next question >

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:

- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

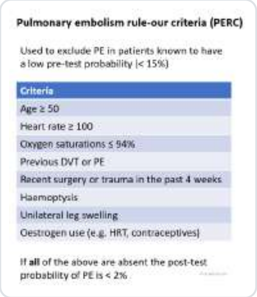
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
    - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

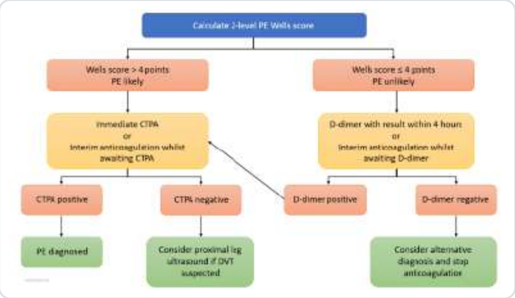
Clinical feature	**Points**
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3

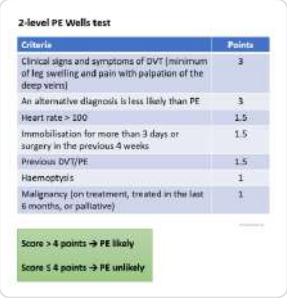
Clinical feature	**Points**
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores
- PE likely - more than 4 points
- PE unlikely - 4 points or less

If a PE is 'likely' (more than 4 points)
- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
    - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)
- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
    - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points
PE likely
Wells score ≤ 4 points
PE unlikely
Immediate CTPA
or
Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours
or
Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

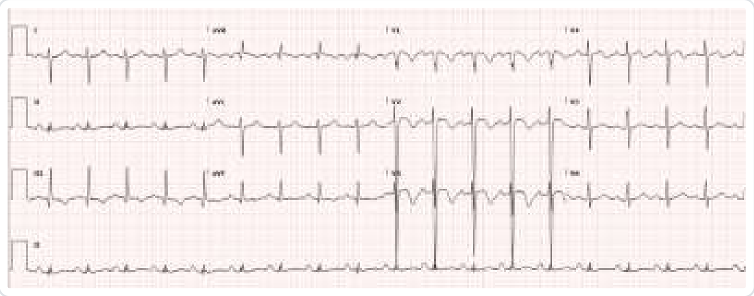
Some other points

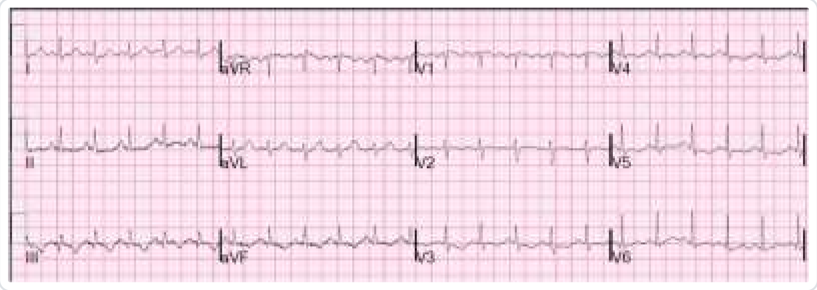
D-dimers

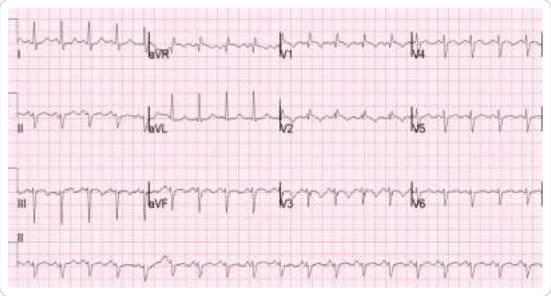
- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen







Chest x-ray

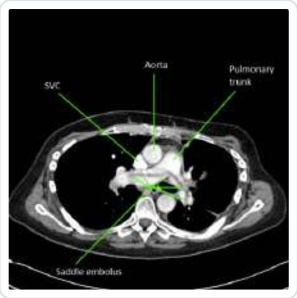
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

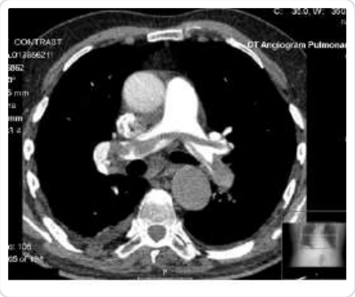
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus


CONTRAST
CT Angiogram Pulmona

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 13 👎 10

The ECG in Pulmonary Embolism

MDcalc 👍 15 👎 8

Wells' Criteria for Pulmonary Embolism

MDcalc 👍 17 👎 8

PERC Rule for Pulmonary Embolism

NICE 👍 20 👎 9

2020 Venous thromboembolism guidelines

Suggest link Report broken link

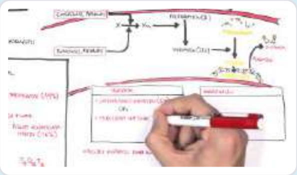
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

Report broken media

Score: **16%**

1	✗
2	✗
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23	✗
24	✗
25	✗

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Question 26 of 328

A 72-year-old man presents to the emergency department with palpitations. An ECG confirms atrial fibrillation. His CHA2DS2-VASc score is 2 and he is commenced on bisoprolol and edoxaban.

He is referred and attends for catheter ablation, which he undergoes successfully. Post-procedure ECG confirms sinus rhythm.

He is followed up 4 weeks later in the clinic and a repeat ECG confirms the maintenance of sinus rhythm.

What is the best course of action at this stage?

- ○ Continue edoxaban for a further 4 weeks then stop ×
- ○ Continue edoxaban for a further 8 weeks then stop ×
- ○ Continue edoxaban long-term ×
- ○ Discontinue edoxaban ×
- ○ Switch to aspirin ×

Submit answer

Reference ranges

Score: **16%**

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A 72-year-old man presents to the emergency department with palpitations. An ECG confirms atrial fibrillation. His CHA2DS2-VASc score is 2 and he is commenced on bisoprolol and edoxaban.

He is referred and attends for catheter ablation, which he undergoes successfully. Post-procedure ECG confirms sinus rhythm.

He is followed up 4 weeks later in the clinic and a repeat ECG confirms the maintenance of sinus rhythm.

What is the best course of action at this stage?

- Continue edoxaban for a further 4 weeks then stop 20%
- Continue edoxaban for a further 8 weeks then stop 10%
- Continue edoxaban long-term 39%
- Discontinue edoxaban 27%
- Switch to aspirin 4%

Patients who've had a catheter ablation for atrial fibrillation still require long-term anticoagulation as per their CHA_2DS_2-VASc score

Important for me Less important

The correct answer is **continue edoxaban long-term**. Although the ablation has been successful and rhythm control has been achieved, patients in this setting have the same stroke risk as those remaining in atrial fibrillation. Anticoagulation should, therefore, be continued long-term.

Continue edoxaban for a further 4 weeks then stop is incorrect.

Continue edoxaban for a further 8 weeks then stop is incorrect.

Discontinue edoxaban is incorrect.

Switch to aspirin is incorrect.

👍 👎 Discuss (4) Improve

Next question >

Atrial fibrillation: rate control and maintenance of sinus rhythm

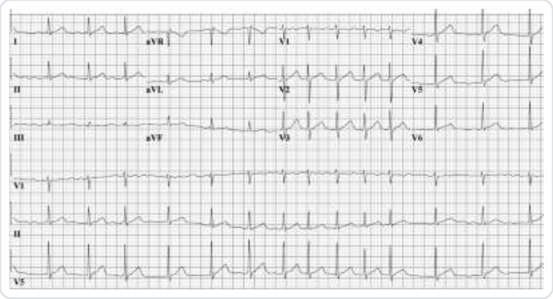
NICE updated its guidelines on the management of atrial fibrillation (AF) in 2021. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines

Patients presenting acutely with AF

If a patient has signs of haemodynamic instability (e.g. hypotension, heart failure) they should be electrically cardioverted, as per the peri-arrest tachycardia guidelines.

For haemodynamically stable patients, the management depends on how acute the AF is:

- < 48 hours: rate or rhythm control
- ≥ 48 hours or uncertain (e.g. patient not sure when symptoms started): rate control
    - if considered for long-term rhythm control, delay cardioversion until they have been maintained on therapeutic anticoagulation for a minimum of 3 weeks



Rate control

Rate control should be offered as the first-line treatment strategy for atrial fibrillation except in people:
- whose atrial fibrillation has a reversible cause
- who have heart failure thought to be primarily caused by atrial fibrillation
- with new-onset atrial fibrillation (< 48 hours)
- with atrial flutter whose condition is considered suitable for an ablation strategy to restore sinus rhythm
- for whom a rhythm-control strategy would be more suitable based on clinical judgement

Medications

Agents used to control rate in patients with atrial fibrillation
- beta-blockers
  - a common contraindication for beta-blockers is asthma
- calcium channel blockers
- digoxin

+ not considered first-line anymore as they are less effective at controlling the heart rate during exercise
- should only be considered if he person does no or very little physical exercise or other rate-limiting drug options are ruled out because of comorbidities
- may have a role if there is coexistent heart failure

Rhythm control

Agents used to maintain sinus rhythm in patients with a history of atrial fibrillation
- beta-blockers
- dronedarone: second-line in patients following cardioversion
- amiodarone: particularly if coexisting heart failure

Catheter ablation

NICE recommends the use of catheter ablation for those with AF who have not responded to or wish to avoid, antiarrhythmic medication.

Technical aspects

- the aim is to ablate the faulty electrical pathways that are resulting in atrial fibrillation. This is typically due to aberrant electrical activity between the pulmonary veins and left atrium
- the procedure is performed percutaneously, typically via the groin
- both radiofrequency (uses heat generated from medium frequency alternating current) and cryotherapy can be used to ablate the tissue

Anticoagulation

- should be used 4 weeks before and during the procedure
- it should be remember that catheter ablation controls the rhythm but does not reduce the stroke risk, even if patients remain in sinus rhythm. Therefore, patients still require anticoagulation as per their CHA$_2$DS$_2$-VASc score
  - if score = 0: 2 months anticoagulation recommended
  - if score > 1: longterm anticoagulation recommended

Outcome

- notable complications include
  - cardiac tamponade
  - stroke
  - pulmonary vein stenosis
- success rate
  - around 50% of patients experience an early recurrence (within 3 months) of AF that often resolves spontaneously
  - longer term, after 3 years, around 55% of patients who've had a single procedure remain in sinus rhythm. Of patients who've undergone multiple procedures around 80% are in sinus rhythm

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 27 👎 19

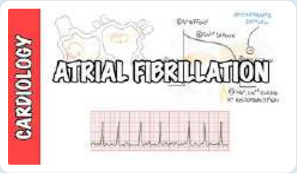
2021 Atrial fibrillation guidelines

European Society of Cardiology 👍 20 👎 9

2020 Atrial fibrillation guidelines

Suggest link Report broken link

Media



Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **15.4%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

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A 43-year-old lady with a history of schizophrenia presents to the emergency department with palpitations, headaches and dizziness for 3 days. She says that she can feel her heart pounding after which she becomes dizzy and feels faint. She has noted these bouts about three to four times per day. She does not suffer from diabetes and is not hypertensive.

Regarding her headaches, she has had them for a long period of time and is quite convinced that there is some sort of sinister problem with her brain, although her doctors do not believe so. She has a younger sister who has had epilepsy for the last 15 years.

She was diagnosed as having a UTI 4 days ago which is currently being treated with ciprofloxacin. Her medication history includes olanzapine and occasional paracetamol for her headaches.

On examination, her pulse was 135 bpm with an irregular rhythm. Blood pressure was 90/60mmHg. Her systemic examination was unremarkable.

Attachment to a cardiac monitor revealed runs of ill sustained polymorphic tachycardia. She was immediately given IV lidocaine to which there was no response.

Which is the most appropriate next step in management?

- ○ Immediate DC cardioversion
- ○ IV amiodarone
- ○ IV flecainide
- ○ IV magnesium sulphate
- ○ IV labetalol

Submit answer

Question 27 of 328

A 43-year-old lady with a history of schizophrenia presents to the emergency department with palpitations, headaches and dizziness for 3 days. She says that she can feel her heart pounding after which she becomes dizzy and feels faint. She has noted these bouts about three to four times per day. She does not suffer from diabetes and is not hypertensive.

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On examination, her pulse was 135 bpm with an irregular rhythm. Blood pressure was 90/60mmHg. Her systemic examination was unremarkable.

Attachment to a cardiac monitor revealed runs of ill sustained polymorphic tachycardia. She was immediately given IV lidocaine to which there was no response.

Which is the most appropriate next step in management?

Immediate DC cardioversion	31%
IV amiodarone	7%
IV flecainide	2%
IV magnesium sulphate	60%
IV labetalol	0%

The scenario above is an acquired long QT syndrome secondary to both ciprofloxacin and olanzapine. Patients with long QT syndromes develop syncope and palpitations as a result of polymorphic ventricular tachycardia (torsades de pointes). Episodes may terminate spontaneously which is usual, but may also evolve into fatal ventricular fibrillation. The corrected QT interval in between the arrhythmia is usually >0.5s.

👍 👎 Discuss (10) Improve

Next question >

Long QT syndrome

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) • methadone • chloroquine • terfenadine** • erythromycin • haloperidol • ondanestron	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Features
- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management
- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

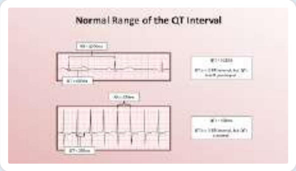
2010 Drug-Induced Long QT syndrome

Life in the Fast Lane 👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link Report broken link

Media



Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **14.8%**

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Question 28 of 328

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (32%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 64/min with a QRS of 136 msec and left bundle branch block. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	128 g/L	Male: (135-180) Female: (115 - 160)
Platelets	335 * 10^9/L	(150 - 400)
WBC	6.8 * 10^9/L	(4.0 - 11.0)
Na^+	136 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	126 µmol/L	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

What alteration to this gentleman's management could potentially decrease this gentleman's probability of being re-admitted?

○ Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation

○ Increase ivabradine

○ Increase bumetanide

○ Increase spironolactone

○ Initiate sacubitril/valsartan

Submit answer

Reference ranges ∨

Score: **14.8%**

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He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

What alteration to this gentleman's management could potentially decrease this gentleman's probability of being re-admitted?

Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation	68%
Increase ivabradine	1%
Increase bumetanide	1%
Increase spironolactone	6%
Initiate sacubitril/valsartan	24%

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

Important for me Less important

The European society of cardiology (ESC) guidelines have highlighted the importance of cardiac resynchronisation in patients with heart failure. Evidence exists indicating the importance of implantation of such devices in patients with intraventricular delay with most studies focusing on the impact of left bundle branch block. The stronger evidence related CRT-P in such patients include left bundle branch block (LBBB) and QRS >150 msec; however the presence of LBBB on its own provided that the QRS is longer than 120 msec is associated with improved mortality/morbidity. There is no evidence of benefit from CRT for patients with QRS <120 msec, while the presence of right bundle branch block (RBBB) implies a worse disease state where CRT may not necessarily benefit such patients.

The possibility of altering medications in this case remains attractive however one should be aware of the potential of introducing detriment to the patient in doing so.

This patient is already tolerating a relatively slow heart rate therefore ivabradine could potentially cause further slowing.

He is borderline hypotensive with raised potassium (making the possibility of increasing spironolactone less favourable.

Sacubitril/valsartan remains an option, however the ESC guidelines dictate that in such cases with noted intraventricular delay the first port of call would be the provision of cardiac resynchronisation therapy.

👍 👎 Discuss (11) Improve

Next question >

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy

- for patients with heart failure and wide QRS
- biventricular pacing

- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training

- improves symptoms but not hospitalisation/mortality

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 4

Cardiac resynchronisation therapy for the treatment of heart failure

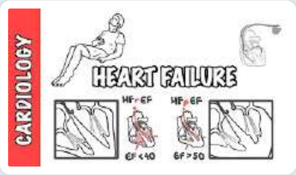
NICE 👍 5 👎 1

2018 Chronic heart failure guidelines

Suggest link

Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

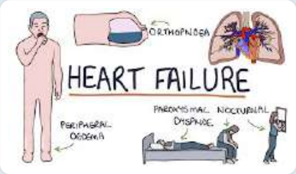
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 0 👎 0

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 4 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 2

Report broken media

Score: **14.3%**

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Question 29 of 328

A 54-year-old presents to his general practitioner with fatigue and shortness of breath. He has no significant past medical history and is on no medications.

Clinical examination reveals a mid-diastolic murmur, louder in expiration, and a loud S1. There is no peripheral oedema. The jugular venous pressure is not raised and chest auscultation is normal. The pulse is regular.

From the options listed, what is the most likely diagnosis?

- ○ Tricuspid stenosis ×
- ○ Moderate mitral stenosis ×
- ○ Mitral regurgitation ×
- ○ Severe mitral stenosis ×
- ○ Mitral valve prolapse ×

Submit answer

Reference ranges

Score: **14.3%**

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From the options listed, what is the most likely diagnosis?

Tricuspid stenosis	6%
Moderate mitral stenosis	63%
Mitral regurgitation	6%
Severe mitral stenosis	19%
Mitral valve prolapse	6%

Mitral stenosis - loud S1

Important for me Less important

Moderate mitral stenosis is the correct answer. This is characterized by a mid-diastolic murmur with a loud S1 heart sound. Left-sided murmurs are louder in expiration. The most common cause is rheumatic fever. This may not always be apparent from the history if it happened in childhood.

Tricuspid stenosis is incorrect. This can also cause a diastolic murmur with a loud S1. However, it will be louder in inspiration as it is a right-sided murmur.

Mitral regurgitation is incorrect. This causes a pansystolic murmur and a soft S1.

Severe mitral stenosis is incorrect. In severe mitral stenosis, the S1 is soft due to lack of pliability and acoustic dampening from a severely calcified valve. Furthermore, there are no obvious clinical features of severe mitral stenosis such as evidence of pulmonary hypertension or right heart failure.

Mitral valve prolapse is incorrect. This murmur may be associated with a 'click' but it is a systolic murmur.

👍 | 👎 | 💬 Discuss (6) | Improve

Next question >

Mitral stenosis ★

Mitral stenosis describes the obstruction of blood flow across the mitral valve from the left atrium to the left ventricle. This leads to increases in pressure within the left atrium, pulmonary vasculature and right side of the heart.

It is said that the causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever. Rarer causes that may be seen in the exam include mucopolysaccharidoses, carcinoid and endocardial fibroelastosis

Features
- dyspnoea
  - ↑ left atrial pressure → pulmonary venous hypertension
- haemoptysis
  - due to pulmonary pressures and vascular congestion
  - may range from pink frothy sputum to sudden haemorrhage secondary to rupture of thin-walled and dilated bronchial veins
- mid-late diastolic murmur (best heard in expiration)
- loud S1
- opening snap
  - indicates mitral valve leaflets are still mobile
- low volume pulse
- malar flush
- atrial fibrillation
  - secondary to ↑ left atrial pressure → left atrial enlargement

Features of severe MS

- length of murmur increases
- opening snap becomes closer to S2

Chest x-ray

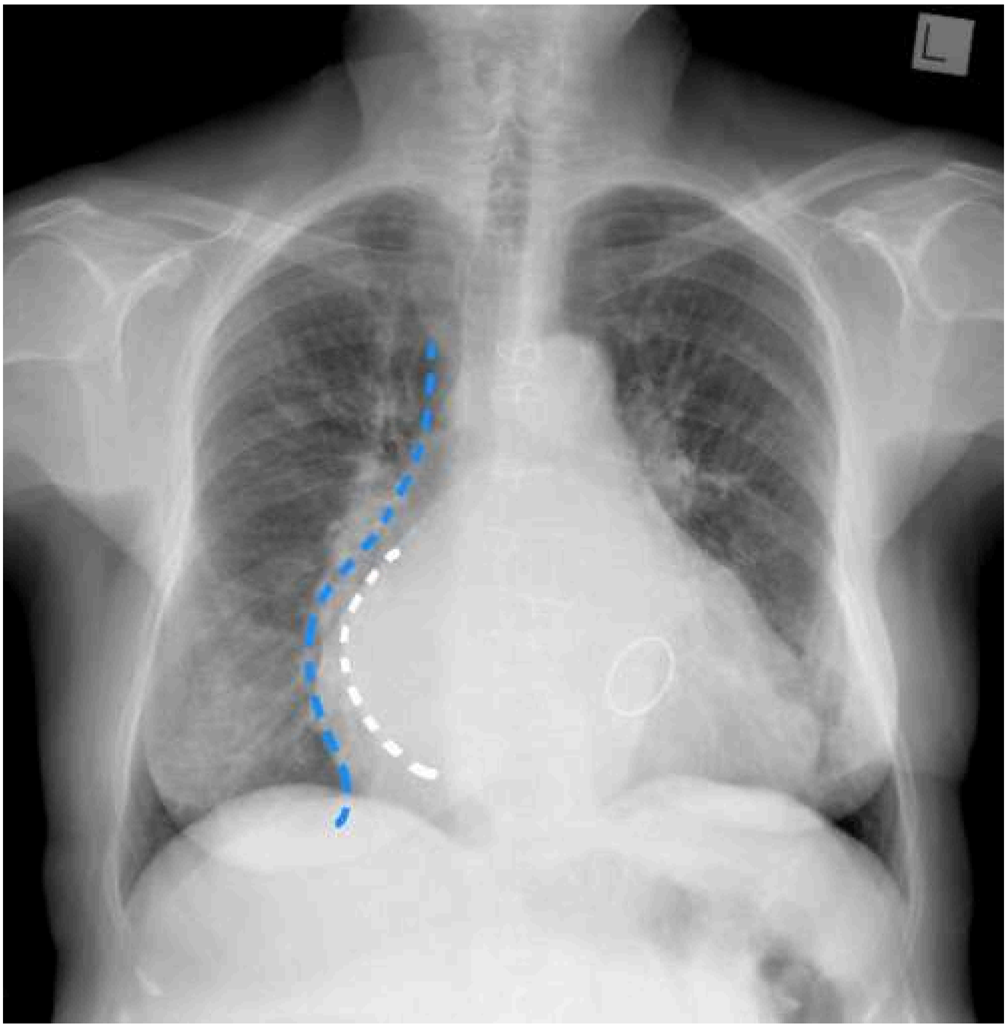
- left atrial enlargement may be seen

Echocardiography

- the normal cross-sectional area of the mitral valve is 4-6 sq cm. A 'tight' mitral stenosis implies a cross-sectional area of < 1 sq cm

Management

- patients with associated atrial fibrillation require anticoagulation
    - currently warfarin is still recommended for patients with moderate/severe MS
    - there is an emerging consensus that direct-acting anticoagulants (DOACs) may be suitable for patients with mild MS who develop atrial fibrillation
- asymptomatic patients
    - monitored with regular echocardiograms
    - percutaneous/surgical management is generally not recommended
- symptomatic patients
    - percutaneous mitral balloon valvotomy
    - mitral valve surgery (commissurotomy, or valve replacement)



© Image used on license from Radiopaedia

Chest x-ray from a patient with mitral stenosis. This patient has had a sternotomy and a prosthetic mitral valve. There is splaying of the carina with elevation of the left main bronchus, a double right heart border and cardiomegaly. The features are those of left atrial enlargement. Although the entire heart is enlarged, a double contour is seen through the right side of the heart. The more medial line is the enlarged left atrium (white dotted line) and the heart border is more lateral (blue dotted line).

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 20 👎 11

Mitral valve stenosis

Suggest link Report broken link

Media

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 4 👎 1

Mitral stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 3 👎 2

Report broken media

Score: **13.8%**

1 ✗
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29	✗

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A 37-year-old lady presents to the Emergency Department with a 4-hour history of sudden-onset chest pain and dyspnoea. Her symptoms started whilst she was watching television. She describes the pain as severe, central and non-radiating. She denies a cough and haemoptysis. Her past medical history is unremarkable and her only regular medication is the oral contraceptive pill.

Examination reveals an overweight lady with a pulse rate of 114/min and a blood pressure of 101/63mmHg. Her respiratory rate is 22 and her oxygen saturations are 94% on air. Her JVP is 5cm but there is no peripheral oedema. Auscultation of the chest reveals scattered crepitations. Her heart sounds are dual with no audible murmurs. A portable chest x-ray shows clear lung fields.

A short time later the patient's blood pressure is recorded at 90/59mmHg. A bedside echocardiogram shows elevated right ventricular filling pressures with evidence of strain. What is the most appropriate management?

- ○ Surgical embolectomy ×
- ○ Alteplase ×
- ○ Unfractionated heparin ×
- ○ Aspirin, clopidogrel, low molecular weight heparin and furosemide ×
- ○ Low molecular weight heparin ×

Submit answer

Reference ranges

← Question 30 of 328 ✗ ⚑ →

A 37-year-old lady presents to the Emergency Department with a 4-hour history of sudden-onset chest pain and dyspnoea. Her symptoms started whilst she was watching television. She describes the pain as severe, central and non-radiating. She denies a cough and haemoptysis. Her past medical history is unremarkable and her only regular medication is the oral contraceptive pill.

Examination reveals an overweight lady with a pulse rate of 114/min and a blood pressure of 101/63mmHg. Her respiratory rate is 22 and her oxygen saturations are 94% on air. Her JVP is 5cm but there is no peripheral oedema. Auscultation of the chest reveals scattered crepitations. Her heart sounds are dual with no audible murmurs. A portable chest x-ray shows clear lung fields.

A short time later the patient's blood pressure is recorded at 90/59mmHg. A bedside echocardiogram shows elevated right ventricular filling pressures with evidence of strain. What is the most appropriate management?

Surgical embolectomy	10%
Alteplase	68%
Unfractionated heparin	4%
Aspirin, clopidogrel, low molecular weight heparin and furosemide	5%
Low molecular weight heparin	13%

This patient has presented with chest pain and dyspnoea in combination with tachycardia, hypotension, and hypoxia. This, in combination with the history of obesity and exogenous oestrogen, makes PE the most likely diagnosis. It is worth noting that PE does not always present with pleuritic chest pain and crepitations and wheeze can often be heard on auscultation.

The management of PE depends on its classification. Intermediate risk (previously 'sub-massive') PE, characterised by the presence of myocardial injury or right ventricular dysfunction, is treated with low molecular weight heparin. High risk (previously 'massive') PE is characterised by shock or hypotension and is best treated by thrombolysis. The patient in this vignette has become hypotensive and would thus benefit from thrombolytic therapy.

Surgical embolectomy is an effective treatment for massive PE but only a few centres are able to offer this. Consequently, thrombolysis remains the preferred first-line treatment for massive PE.

Acute MI with cardiogenic shock would be a key differential, but the clear lung fields on chest x-ray and lack of peripheral oedema go against this. Therefore, treatment with aspirin and clopidogrel would not be beneficial.

👍 👎 Discuss (11) Improve

Next question >

Pulmonary embolism: management ★

NICE updated their guidelines on the management of venous thromboembolism (VTE) in 2020. Some of the key changes include recommending the following:

- the use of direct oral anticoagulants (DOACs) as first-line treatment for most people with VTE
- the use of DOACs in patients with active cancer, as opposed to low-molecular weight heparin as was the previous recommendation
- outpatient treatment in low-risk pulmonary embolism (PE) patients
- routine cancer screening is no longer recommended following a VTE diagnosis

Outpatient treatment in low-risk PE patients

Deep vein thrombosis has for a long time been treated on an outpatient condition. In contrast, patients with any form of PE were typically admitted. However, in recent years patients with a new diagnosis of PE who are deemed low-risk are now increasingly managed as outpatients. NICE formally supported this approach in their latest guidance.

- NICE recommends using a 'validated risk stratification tool' to determine the suitability of outpatient treatment.
    - no guidance is given as to what tool should be used
    - the 2018 British Society guidelines support the use of the Pulmonary Embolism Severity Index (PESI) score
- key requirements would clearly be haemodynamic stability, lack of comorbidities and support at home

Anticogulant therapy

The cornerstone of VTE management is anticoagulant therapy. This was historically done with warfarin, often preceded by heparin until the INR was stable. However, the development of DOACs, and an evidence base supporting their efficacy, has changed modern management.

Choice of anticoagulant

- the big change in the 2020 guidelines was the increased use of DOACs
- apixaban or rivaroxaban (both DOACs) should be offered first-line following the diagnosis of a PE
    - instead of using low-molecular weight heparin (LMWH) until the diagnosis is confirmed, NICE now advocate using a DOAC once a diagnosis is suspected, with this continued if the diagnosis is confirmed
    - if neither apixaban or rivaroxaban are suitable then either LMWH followed by dabigatran or edoxaban OR LMWH followed by a vitamin K antagonist (VKA, i.e. warfarin)
- if the patient has active cancer
    - previously LMWH was recommended
    - the new guidelines now recommend using a DOAC, unless this is contraindicated
- if renal impairment is severe (e.g. < 15/min) then LMWH, unfractionated heparin or LMWH followed by a VKA
- if the patient has antiphospholipid syndrome (specifically 'triple positive' in the guidance) then LMWH followed by a VKA should be used

Length of anticoagulation

- all patients should have anticoagulation for at least 3 months
- continuing anticoagulation after this period is partly determined by whether the VTE was provoked or unprovoked
    - a provoked VTE is due to an obvious precipitating event e.g. immobilisation following major surgery. The implication is that this event was transient and the patient is no longer at increased risk
    - an unprovoked VTE occurs in the absence of an obvious precipitating event, i.e. there is a possibility that there are unknown factors (e.g. mild thrombophilia) making the patient more at risk from further clots
- if the VTE was provoked the treatment is typically stopped after the initial 3 months (3 to 6 months for people with active cancer)
- if the VTE was unprovoked then treatment is typically continued for up to 3 further months (i.e. 6 months in total)
    - NICE recommend that whether a patient has a total of 3-6 months anticoagulant is based upon balancing a person's risk of VTE recurrence and their risk of bleeding
    - the ORBIT score can be used to help assess the risk of bleeding
    - NICE state: '*Explain to people with unprovoked DVT or PE and a low bleeding risk that the benefits of continuing anticoagulation treatment are likely to outweigh the risks.* '. The implication of this is that in the absence of a bleeding risk factors, patients are generally better off continuing anticoagulation for a total of 6 months

PE with haemodynamic instability

Thrombolysis

- thrombolysis is now recommended as the first-line treatment for massive PE where there is circulatory failure (e.g. hypotension)
- other invasive approaches should be considered where appropriate facilities exist

Patients who have repeat pulmonary embolisms, despite adequate anticoagulation, may be considered for inferior vena cava (IVC) filters. These work by stopping clots formed in the deep veins of the leg from moving to the pulmonary arteries. IVC filter use is currently supported by NICE but other studies suggest a weak evidence base - please see the link for more details.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 4 👎 7

2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism

Clinical Knowledge Summaries 👍 8 👎 8

Anticoagulation guidelines

NICE 👍 14 👎 9

2020 Venous thromboembolism guidelines

British Committee for Standards in Haematology 👍 10 👎 6

Guidelines on oral anticoagulation (warfarin): fourth edition - 2011 update

British Thoracic Society 👍 10 👎 5

2018 Initial outpatient management of pulmonary embolism guidelines

Journal of the American College of Cardiology 👍 13 👎 5

2017 Inferior Vena Cava Filters to Prevent Pulmonary Embolism

Suggest link

Report broken link

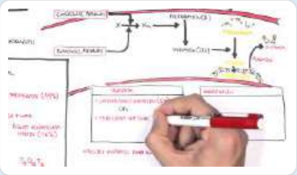
Media

Pulmonary embolism

Osmosis - YouTube 👍 0 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 0 👎 0



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **13.3%**

1 ✗

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28	✗
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30	✗

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Back to top

A 58-year-old man attends cardiology clinic with retrosternal chest pain on exertion. He reports the onset of his symptoms when walking more than 50 metres on flat ground or when playing on the floor with his grandchildren. The symptoms have been present at the current level for at least the previous 9 months and the patient denied any episodes of pain at rest. The patients GP has previously attempted treatment with bisoprolol and amlodipine but both medications were discontinued for unwanted effects (bradycardia and ankle swelling respectively). The patient has no other medical history except for hypercholesterolaemia. Regular medications are aspirin 75 mg daily and pravastatin 10 mg daily with nitrate spray and sildenafil used as required. He is formerly a heavy smoker but managed to quit the previous year.

Clinical examination is unremarkable with no evidence of cardiac failure. Blood pressure is 102 / 72 mmHg.

Please see below for the results of previous investigations.

Electrocardiogram: sinus rhythm at 58 beats per minute; borderline left axis deviation; normal QRS complex; non-specific lateral ST segment abnormalities; normal T waves.

Transthoracic echocardiogram: normal valvular function; no regional wall motion abnormality; ejection fraction 55-60 %.

Urea	6.7 mmol / L
Creatinine	80 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L

Cardiac stress magnetic resonance imaging: significant evidence of ischaemia in the region of the lateral left ventricle; estimated 20 % of LV myocardium with significant evidence of ischaemia.

What is the most appropriate management of the patients chest pain?

○ Ticagrelor ×

○ Nicorandil ×

○ Percutaneous coronary intervention ×

○ Ivabradine ×

○ Long-acting isosorbide mononitrate ×

Submit answer

Reference ranges ⌄

Score: **13.3%**

1	✗
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Cardiac stress magnetic resonance imaging: significant evidence of ischaemia in the region of the lateral left ventricle; estimated 20 % of LV myocardium with significant evidence of ischaemia.

What is the most appropriate management of the patients chest pain?

Ticagrelor	2%
Nicorandil	20%
Percutaneous coronary intervention	57%
Ivabradine	6%
Long-acting isosorbide mononitrate	15%

The role of percutaneous coronary intervention in stable angina is a controversial subject. Revascularisation has not been shown to reduce mortality or the rate of myocardial infarction in such patients. However, sub-group analysis of randomised controlled trials suggests some benefit for the above outcomes over medical therapy. Therefore, ESC guidelines recommend offering revascularisation to patients with stable coronary artery disease and ischaemia in > 10 % of the left ventricle. Also, the patient's age and lack of co-morbidities make this the most appropriate strategy in this case.

The other possible answers that are options for the medical management of stable angina are contraindicated in this patient. Both nicorandil and ISMN are contra-indicated for this patient due to his borderline hypotension and use of sildenafil. Ivabradine is contra-indicated due to the patient's history of bradycardia. It is also not used in patients with moderate to severe angina symptoms as has been shown to increase the rate of cardiovascular events in such patients.

Ticagrelor is a P2Y12 adenosine diphosphate receptor blocker with utility as an oral antiplatelet agent.

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stunting in stable angina? BMJ 2016;352:i205.

👍 👎 Discuss (9) Improve

Next question >

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
  - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
  - a long-acting nitrate
  - ivabradine
  - nicorandil
  - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance

- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

Suggest link

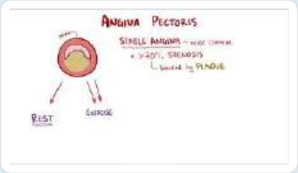
Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **16.1%**

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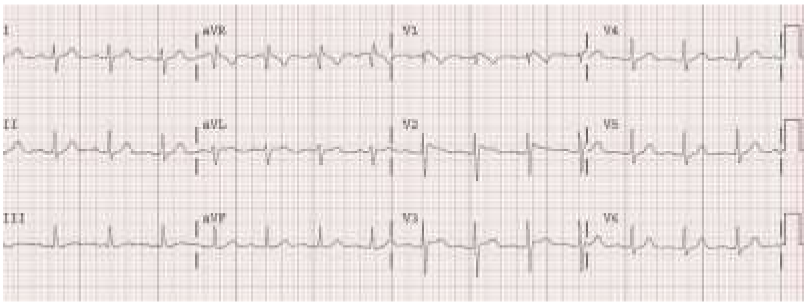
A 30-year-old man is admitted to the Emergency Department after suffering a 'blackout' whilst at work. His colleagues report him collapsing without warning whilst waiting at the water machine. There has never happened before and he is normally fit and well.

On examination blood pressure is 102/68 mmHg, pulse 88/min, oxygen saturations 99% on room air and respiratory rate 16/min.

An ECG is taken:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

- ○ Hypertrophic obstructive cardiomyopathy
- ○ Anterior myocardial infarction
- ○ Long QT syndrome type 1
- ○ Arrhythmogenic right ventricular dysplasia
- ○ Brugada syndrome

Submit answer

Reference ranges

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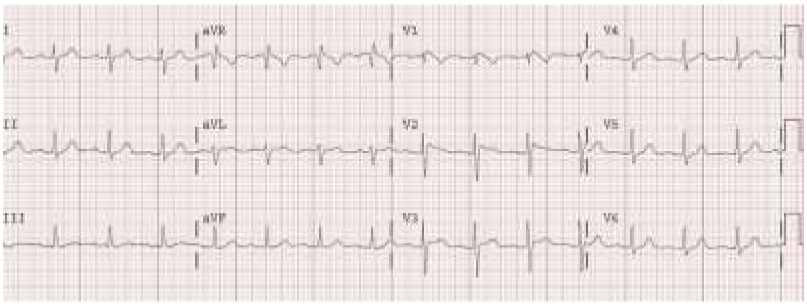


© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

- Hypertrophic obstructive cardiomyopathy 6%
- Anterior myocardial infarction 1%
- Long QT syndrome type 1 7%
- Arrhythmogenic right ventricular dysplasia 19%
- Brugada syndrome 67%

The ECG shows a Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave.

Discuss (5) Improve

Next question

Brugada syndrome ★

Brugada syndrome is a form of inherited cardiovascular disease with may present with sudden cardiac death. It is inherited in an autosomal dominant fashion and has an estimated prevalence of 1:5,000-10,000. Brugada syndrome is more common in Asians.

Pathophysiology

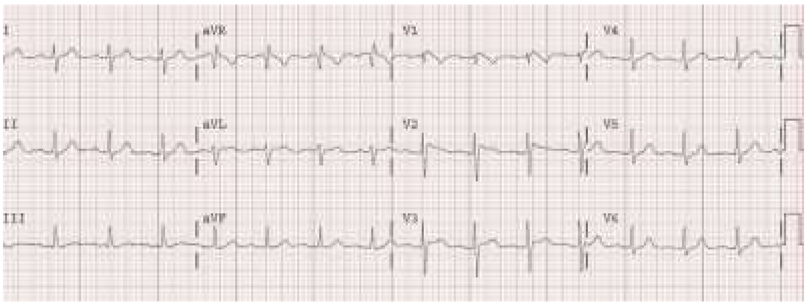
- a large number of variants exist
- around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the myocardial sodium ion channel protein

ECG changes

- convex ST segment elevation > 2mm in > 1 of V1-V3 followed by a negative T wave
- partial right bundle branch block
- the ECG changes may be more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases of Brugada syndrome

Management

- implantable cardioverter-defibrillator



© Image used on license from Dr Smith, University of Minnesota

ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

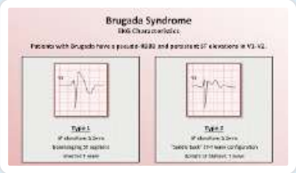
Links

Life in the fast lane 👍 29 👎 5

Brugada syndrome

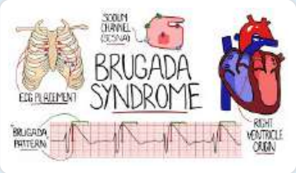
Suggest link Report broken link

Media


Brugada Syndrome
EKG Characteristics
Type 1
Type 2

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 12 👎 0


SODIUM CHANNEL (SCN5A)
BRUGADA SYNDROME
ECG PLACEMENT
BRUGADA PATTERN
RIGHT VENTRICLE ORIGIN

Brugada syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **18.8%**

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27	✗
28	✗
29	✗
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31	✓
32	✓

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Question 33 of 328

A 66-year-old gentleman presented to the Emergency Department complaining of severe abdominal pain for the last day. The pain occurred immediately after he ate, and he described it as an excruciating pain in the central of his abdomen. It lessened during periods of starvation, and a few hours ago passed dark red blood within his stools on one occasion, leading to his admission. His bowels were otherwise normal and prior to the pain had been relatively well in himself. In addition, he had been feeling unwell for the last couple of days, complaining of night sweats and tiredness. His past medical history comprised angina which had been confirmed three days ago upon undergoing a coronary angiogram which demonstrated diffuse triple vessel disease. In addition, he had a past medical history of hypertension, hypercholesterolaemia and COPD. He smoked 30 cigarettes per day for 25 years and did not consume alcohol and was taking amlodipine 5mg OD, ramipril 7.5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg OD, beclometasone 200mcg BD & salbutamol PRN.

Examination revealed an obese gentleman with a temperature of 37.6ºC, a heart rate of 118bpm, respiratory rate of 18/min and blood pressure of 142/82 mmHg. Examination of his cardiovascular and respiratory systems revealed the absence of his pedal pulses bilaterally, with no evidence of ulceration or gangrene. A purpuric rash was noted on both halluxes.

Initial investigations revealed the following results:

Hb	122 g/l
Platelets	132 * 10^9/l
WBC	11.2 * 10^9/l
Eosinophils	0.6 * 10^9/l
ESR	52 mm/hr

Na$^+$	136 mmol/l
K$^+$	5.8 mmol/l
Urea	13.3 mmol/l

Creatinine	158 µmol/l
CRP	38 mg/l

Urinalysis protein +, otherwise nil else

ECG: heart rate 118bpm sinus tachycardia, Q waves II/III/aVR

Blood results 4 weeks ago (done by GP when titrated ramipril)

Na^+	142 mmol/l
K^+	4.8 mmol/l
Urea	6.6 mmol/l
Creatinine	86 µmol/l

Which one of the following is the most likely diagnosis?

○ Contrast induced nephropathy ×

○ Cholesterol atheroemboli ×

○ Polyarteritis nodosa ×

○ Infective endocarditis ×

○ Microscopic polyangiitis ×

Submit answer

Reference ranges ⌄

Score: **18.8%**

1 ✗
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Question 33 of 328

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Na^+	142 mmol/l
K^+	4.8 mmol/l
Urea	6.6 mmol/l
Creatinine	86 µmol/l

Which one of the following is the most likely diagnosis?

Contrast induced nephropathy	5%
Cholesterol atheroemboli	75%
Polyarteritis nodosa	13%
Infective endocarditis	2%
Microscopic polyangiitis	5%

Cholesterol atheroemboli may occur as a consequence of manipulation of arteries lined with atheromatous plaques, such as a result of angiography. Cholesterol emboli may involve multiple organs, as a result of widespread dissemination. Signs and symptoms often involve a low-grade inflammatory response with fever and malaise. Cutaneous emboli lodging may affect the distal extremities (as is evident here by a vasculitic rash). Organs involved include the respiratory system, gastrointestinal system (as is manifest by mesenteric ischaemia here) and the renal system resulting in renal failure, with often proteinuria upon urinalysis. A key differential is contrast nephropathy, but cutaneous involvement and other organ involvement is very uncommon and the renal failure tends to be transient.

Discuss (7) Improve

Next question

Cholesterol embolisation

Overview

- cholesterol emboli may break off causing renal disease
- the majority of cases are secondary to vascular surgery or angiography. Other causes include severe atherosclerosis, particularly in large arteries such as the aorta

Features

- eosinophilia
- purpura
- renal failure
- livedo reticularis

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.2%**

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Question 34 of 328

A 68 year old gentleman with a past medical history of type 2 diabetes mellitus and hypertension attends the Emergency Department complaining of general malaise and feeling unwell. He had undergone successful primary coronary intervention (PCI) post myocardial infarction 5 weeks previously. He has a temperature of 38.2 degrees Celsius, blood pressure 164/87 mmHg and the doctor notes a lacy reticular rash extending over his legs.

Hb	12.2g/dl
Eosinophils	1.2 * 10^9/l
WBC	14.5 * 10^9/l
Urea	8mmol/l
Creatinine	142µmol/l

What is the most likely cause of his symptoms?

- ○ Allergic rash to contrast ×
- ○ Infected femoral site ×
- ○ Cholesterol embolus ×
- ○ Infective endocarditis ×
- ○ Polyarteritis nodosa ×

Submit answer

Reference ranges

Score: **18.2%**

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Question 34 of 328

A 68 year old gentleman with a past medical history of type 2 diabetes mellitus and hypertension attends the Emergency Department complaining of general malaise and feeling unwell. He had undergone successful primary coronary intervention (PCI) post myocardial infarction 5 weeks previously. He has a temperature of 38.2 degrees Celsius, blood pressure 164/87 mmHg and the doctor notes a lacy reticular rash extending over his legs.

Hb	12.2g/dl
Eosinophils	1.2 * 10^9/l
WBC	14.5 * 10^9/l
Urea	8mmol/l
Creatinine	142µmol/l

What is the most likely cause of his symptoms?

Allergic rash to contrast	1%
Infected femoral site	1%
Cholesterol embolus	90%
Infective endocarditis	4%
Polyarteritis nodosa	4%

This is a classic story for a cholesterol embolus post an invasive arterial procedure, whereby a plaque has been ruptured and there is lodging of debris in small/medium arteries causing mechanical occlusion, inflammation and end-organ damage. Patients may have the lacy reticular rash of livedo reticularlaris and this, together with acute renal failure and an eosinophilia should lead the clinician to consider this diagnosis.

Patients tend to present with non-specific symptoms such as fever, weight loss and myalgia for weeks to months before developing end-organ damage or stroke.

The current mainstay of treatment is supportive.

Discuss (7) | Improve

Next question >

Cholesterol embolisation

Overview
- cholesterol emboli may break off causing renal disease
- the majority of cases are secondary to vascular surgery or angiography. Other causes include severe atherosclerosis, particularly in large arteries such as the aorta

Features
- eosinophilia
- purpura
- renal failure
- livedo reticularis

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **17.6%**

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Question 35 of 328

A 70-year-old man is admitted to hospital with chest pain associated with nausea and vomiting. There was no recent history of shortness of breath or wheeze. ECG demonstrated inferolateral ST depression and troponin was significantly elevated at 12 hours after symptom onset.

His past medical history included chronic obstructive pulmonary disease, hypertension and a previous MI 3 years previously. The patient reported two exacerbations of his COPD within the last 18 months, neither of which required hospital admission. He had never required intubation or non-invasive ventilation due to his COPD.

Regular medications included bendroflumethiazide 2.5 mg OD, inhaled tiotropium 18 microg OD and inhaled salbutamol 100 microg PRN. There were no known drug allergies. The patient was a retired engineer, an ex-smoker and lived independently with his wife.

The initial impression was of non-ST elevation myocardial infarction and treatment was initiated with aspirin, clopidogrel, fondaparinux, ramipril and atorvastatin. The patient subsequently mobilised pain-free on the ward and was discharged home with a plan for outpatient stress echocardiogram.

Recent pulmonary function tests are given in the table below.

Forced vital capacity	105 % predicted
Forced expiratory volume (1s)	67 % predicted
FVC / FEV1	64 % predicted

Transthoracic echocardiogram: no valvular abnormality; mild-moderate systolic impairment of lateral left ventricle; normal right ventricular function

What is the most appropriate plan for beta-blockade therapy for this patient?

○ Low dose bisoprolol with slow up-titration of dose

○ Low dose carvedilol with slow up-titration of dose

○ Low dose bisoprolol

○ Low dose carvedilol

○ Beta-blockade therapy inappropriate

Submit answer

Reference ranges ∨

Score: **17.6%**

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FVC / FEV1	64 % predicted

Transthoracic echocardiogram: no valvular abnormality; mild-moderate systolic impairment of lateral left ventricle; normal right ventricular function

What is the most appropriate plan for beta-blockade therapy for this patient?

Low dose bisoprolol with slow up-titration of dose	40%
Low dose carvedilol with slow up-titration of dose	23%
Low dose bisoprolol	9%
Low dose carvedilol	6%
Beta-blockade therapy inappropriate	22%

NICE guidance on secondary prevention following myocardial infarction includes a recommendation for beta-blocker therapy as it has been shown to reduce mortality. In patients with COPD or asthma, beta-blockers have sometimes been held due to concerns abut precipitating bronchospasm. However, a large observational study has shown substantial survival benefits for patients with COPD who have suffered a myocardial infarction in starting beta-blockers during hospital admission (adjusted hazard ratio 0.50).

Therefore, beta-blocker therapy is indicated in this patient. The most appropriate choice is the cardio-selective agent (bisoprolol) started at a low dose and slowly up-titrated as per current guidance.

Qunit J, Herrett E, Bhaskaran K, Timmis A, Hemingway H, Wedzicha J et al. Effect of beta-blockers on mortality after myocardial infarction in adults with COPD: population-based cohort study of UK electronic healthcare records. BMJ 2013;347:f6650.

👍 👎 Discuss (13) Improve

Next question ›

Beta-blockers ★

Beta-blockers are an important class of drug used mainly in the management of cardiovascular disorders.

Indications

- angina
- post-myocardial infarction
- heart failure: beta-blockers were previously avoided in heart failure but there is now strong evidence that certain beta-blockers improve both symptoms and mortality
- arrhythmias: beta-blockers have now replaced digoxin as the rate-control drug of choice in atrial fibrillation
- hypertension: the role of beta-blockers has diminished in recent years due to a lack of evidence in terms of reducing stroke and myocardial infarction.
- thyrotoxicosis
- migraine prophylaxis
- anxiety

Examples
- atenolol
- propranolol: one of the first beta-blockers to be developed. Lipid soluble therefore crosses the blood-brain barrier

Side-effects
- bronchospasm
- cold peripheries
- fatigue
- sleep disturbances, including nightmares
- erectile dysfunction

Contraindications
- uncontrolled heart failure
- asthma
- sick sinus syndrome
- concurrent verapamil use: may precipitate severe bradycardia

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 7

Beta-blockers

Suggest link Report broken link

Media

Adrenergic Antagonists

Alpha and beta blockers

Speed Pharmacology - YouTube 👍 2 👎 1

Report broken media

Score: **17.1%**

1 ✗
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Question 36 of 328

A 34-year-old Afro-Caribbean female presents to the heart failure clinic and would like your advice. Two years ago, she presented with shortness of breath and reduced exercise tolerance three months after her second pregnancy. A chest x-ray demonstrated bilateral alveolar shadowing. The symptoms were improved following diuresis and she remains on an ACE inhibitor, beta blocker and furosemide. She continued to be followed up with echocardiograms, her last demonstrating LV EF of 30%. She would like to try for a third pregnancy. What would you recommend regarding pregnancy?

- ○ Pregnancy is advised as normal ×
- ○ Pregnancy is not advised ×
- ○ Pregnancy is advised with increased ACEi ×
- ○ Pregnancy is advised with monthly echocardiograms ×
- ○ Pregnancy is advised with increased beta blockade ×

Submit answer

Reference ranges

Score: **17.1%**

1 ✗
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Question 36 of 328 ✗

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Pregnancy is advised as normal	2%
Pregnancy is not advised	83%
Pregnancy is advised with increased ACEi	1%
Pregnancy is advised with monthly echocardiograms	11%
Pregnancy is advised with increased beta blockade	3%

The patient presented with peripartum cardiomyopathy, defined as LV ejection fraction reduction in the last month or within 5 months of giving birth, particularly prevalent in black women. The key in this question is whether LV dysfunction was persistent. The last echo demonstrates persistent reduced EF and in this case, pregnancy is NOT advised due to the risk of LV failure progression and significant mortality risk of up to 20%[1]. Those with recovered LV function are likely to also experience deterioration in LV function but pregnancy is not absolutely contraindicated.

Treatment is similar to that of heart failure, consisting of beta blockade, diuretics but avoidance of ACEi due to the risk to the foetus or breast-feeding baby. Digoxin may also have a greater role in PPCM than in classic heart failure. The degree of residual LV dysfunction is difficult to predict but a number of factors appear to be predictive of worse outcomes, namely LVEF <30% at presentation, raised troponin I and LV end-diastolic diameter > 6cm.

1. Elkayam U, Tummala PP, Rao K et al. Maternal and fetal outcomes of subsequent pregnancies in women with peripartum cardiomyopathy. N Engl J Med. 2001;344(21):1567

👍 👎 Discuss Improve

Next question >

Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is the most common form of cardiomyopathy, accounting for 90% of cases.

Causes:

- idiopathic: the most common cause
- myocarditis: e.g. Coxsackie B, HIV, diphtheria, Chagas disease
- ischaemic heart disease
- peripartum
- hypertension
- iatrogenic: e.g. doxorubicin
- substance abuse: e.g. alcohol, cocaine
- inherited: either a familial genetic predisposition to DCM or a specific syndrome e.g. Duchenne muscular dystrophy
    - around a third of patients with DCM are thought to have a genetic predisposition
    - a large number of heterogeneous defects have been identified
    - the majority of defects are inherited in an autosomal dominant fashion although other patterns of inheritance are seen
- infiltrative e.g. haemochromatosis, sarcoidosis

+ these causes may also lead to restrictive cardiomyopathy

- nutritional e.g. wet beriberi (thiamine deficiency)

Pathophysiology

- dilated heart leading to predominately systolic dysfunction
- all 4 chambers are dilated, but the left ventricle more so than right ventricle
- eccentric hypertrophy (sarcomeres added in series) is seen

Features

- classic findings of heart failure
- systolic murmur: stretching of the valves may result in mitral and tricuspid regurgitation
- S3
- 'balloon' appearance of the heart on the chest x-ray

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

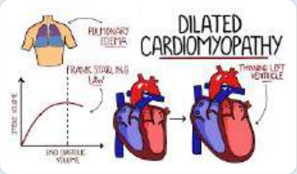
Media

CARDIOLOGY
DILATED CARDIOMYOPATHY

Dilated cardiomyopathy

Armando Hasudungan - YouTube

👍 1 👎 0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DILATED CARDIOMYOPATHY
FRANK STARLING LAW
THINNING LEFT VENTRICLE

Dilated cardiomyopathy

Rhesus Medicine - YouTube

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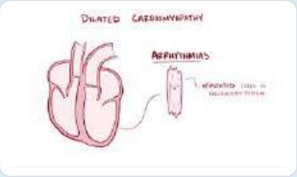
CARDIOLOGY
CARDIOMYOPATHY
OVERVIEW

Cardiomyopathy Overview - types (dilated, hypertrophic, restrictive), pathophysiology and treatment

Armando Hasudungan - YouTube 👍 2 👎 1


DILATED CARDIOMYOPATHY
ARRHYTHMIAS

Dilated cardiomyopathy

Osmosis - YouTube 👍 2 👎 2

Report broken media

Score: **16.7%**

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Question 37 of 328

A 67-year-old woman with mitral regurgitation attends a cardiology clinic for her regular follow-up. She is asymptomatic, can perform daily tasks, and has a New York heart association (NYHA) functional classification of I. On examination, there is a loud rumbling pan-systolic murmur heard at the apex. She is noted to be in atrial fibrillation, which was not previously known. A recent echocardiogram shows an ejection fraction of 62% with mild left atrial dilatation.

Which feature from the history would be the strongest indication for referral for valve replacement?

- ○ Age ×
- ○ Atrial fibrillation ×
- ○ Left atrial dilatation ×
- ○ Loud murmur ×
- ○ Ejection fraction <65% ×

Submit answer

Reference ranges

Score: **16.7%**

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Question 37 of 328 ✓

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Which feature from the history would be the strongest indication for referral for valve replacement?

Age	3%
Atrial fibrillation	59%
Left atrial dilatation	24%
Loud murmur	7%
Ejection fraction <65%	8%

New AF in mitral regurgitation -> refer for mitral valve replacement

Important for me Less important

Surgery is indicated in patients with signs of left ventricular (LV) dysfunction. If LV function is preserved, surgery should be considered in asymptomatic patients with new-onset atrial fibrillation (AF) or pulmonary hypertension. Severe symptomatic mitral regurgitation is also an indication for replacement.

AF develops as a consequence of increased left atrial pressures (due to backflow), resulting in left atrial dilatation. This is, therefore, the strongest indication for replacement.

In the absence of AF, left atrial dilatation only becomes an indication for replacement if it is significant (≥60 mL/m2 body surface area [BSA]).

Age itself is not an indication for mitral valve replacement. Age will be considered alongside other co-morbidities and functional baseline when deciding if she will be suitable for a general anaesthetic for the procedure.

The loudness of a murmur typically doesn't impact the decision to proceed with surgery.

Ejection fraction becomes relevant in asymptomatic patients. 2017 European society of cardiology (ESC) and European association for cardiothoracic surgery (EACTS) guidelines for the management of valvular heart disease suggest that replacement should be considered in asymptomatic patients with an ejection fraction of less than 60%.

👍 👎 💬 Discuss (3) Improve

Next question ›

Mitral regurgitation ★

Also known as mitral insufficiency, mitral regurgitation (MR) occurs when blood leaks back through the mitral valve on systole. It is the second most common valve disease after aortic stenosis. The mitral valve is located between the left atrium and ventricle, and regurgitation leads to a less efficient heart as less blood is pumped through the body with each contraction. This said, MR is common in otherwise healthy patients to a trivial degree and does not need treatment.

As the degree of regurgitation becomes more severe, the body's oxygen demands may exceed what the heart can supply and as a result, the myocardium can thicken over time. While this may be benign initially, patients may find themselves increasingly fatigued as a thicker myocardium becomes less efficient, and eventually go into irreversible heart failure.

Risk factors

- Female sex
- Lower body mass
- Age
- Renal dysfunction

- Prior myocardial infarction
- Prior mitral stenosis or valve prolapse
- Collagen disorders e.g. Marfan's Syndrome and Ehlers-Danlos syndrome

Causes

- Following coronary artery disease or post-MI: if the papillary muscles or chordae tendinae are affected by a cardiac insult, mitral valve disease may ensue as a result of damage to its supporting structures.
- Mitral valve prolapse: Occurs when the leaflets of the mitral valve is deformed so the valve does not close properly and allows for backflow. Most patients with this have a trivial degree of mitral regurgitation.
- Infective endocarditis: When vegetations from the organisms colonising the heart grow on the mitral valve, it is prevented from closing properly. Patients with abnormal valves are more likely to develop endocarditis as opposed to their peers.
- Rheumatic fever: While this is uncommon in developed countries, rheumatic fever can cause inflammation of the valves and therefore result in mitral regurgitation.
- Congenital

Symptoms

- Most patients with MR are asymptomatic, and patients suffering from mild to moderate MR may stay largely asymptomatic indefinitely. Symptoms tend to be due to failure of the left ventricle, arrhythmias or pulmonary hypertension. This may present as fatigue, shortness of breath and oedema.

Signs

- The murmur heard on auscultation of the chest is typically a pansystolic murmur described as 'blowing'. It is heard best at the apex and radiating into the axilla. S1 may be quiet as a result of incomplete closure of the valve. Severe MR may cause a widely split S2

Investigations

- ECG may show a broad P wave, indicative of atrial enlargement
- Cardiomegaly may be seen on chest x-ray, with an enlarged left atrium and ventricle

- Echocardiography is crucial to diagnosis and to assess severity

Treatment options

- Medical management in acute cases involves nitrates, diuretics, positive inotropes and an intra-aortic balloon pump to increase cardiac output
- If patients are in heart failure, ACE inhibitors may be considered along with beta-blockers and spironolactone
- In acute, severe regurgitation, surgery is indicated
- The evidence for repair over replacement is strong in degenerative regurgitation, and is demonstrated through lower mortality and higher survival rates
- When this is not possible, valve replacement with either an artificial valve or a pig valve is considered

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

MITRAL REGURGITATION (INSUFFICIENCY)

Mitral regurgitation

Armando Hasudungan - YouTube

👍 5 👎 0

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 1 👎 0

Mitral regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1

Report broken media

Score: **18.9%**

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An 84-year-old man comes for review. Four weeks ago an opportunistic blood pressure reading was taken and recorded as 150/92 mmHg. You therefore arranged ambulatory blood pressure monitoring (ABPM) along with a standard hypertension work-up. You did not calculate his 10-year cardiovascular risk on account of his age. The following results were obtained:

Na^+	141 mmol/l
K^+	4.2 mmol/l
Urea	6.5 mmol/l
Creatinine	101 µmol/l
Total cholesterol	4.9 mmol/l
HDL cholesterol	1.2 mmol/l
Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg. What is the most appropriate course of action?

- ○ Diagnose stage 1 hypertension and advise about lifestyle changes
- ○ Start treatment with an ACE inhibitor
- ○ Start treatment with a calcium channel blocker
- ○ Start treatment with a thiazide-like diuretic
- ○ Repeat the ABPM

Submit answer

31	✓
32	✓
33	✗
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35	✗
36	✗
37	✓
38	-

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Back to top

Question 38 of 328

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Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg. What is the most appropriate course of action?

- Diagnose stage 1 hypertension and advise about lifestyle changes 58%
- Start treatment with an ACE inhibitor 8%
- Start treatment with a calcium channel blocker 26%
- Start treatment with a thiazide-like diuretic 1%
- Repeat the ABPM 7%

Stage 1 hypertension is defined by an ABPM reading of >= 135/85 mmHg, with stage 2 hypertension having a cut-off of >= 150/95 mmHg.

This patient therefore has stage 1 hypertension. As they are > 80 years they do not need treatment.

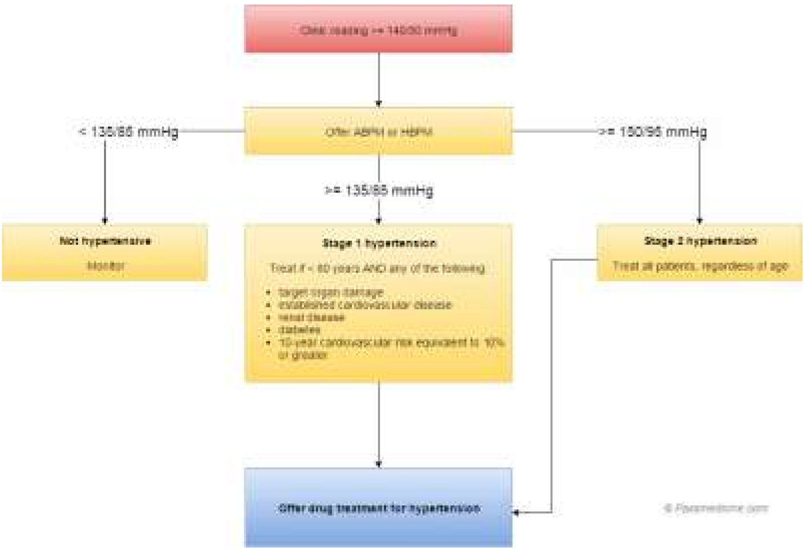
Discuss (5) Improve

Next question >

Hypertension: diagnosis

NICE published updated guidelines for the management of hypertension in 2019. This builds on the significant guidelines released in 2011 that recommended:

- classifying hypertension into stages
- recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Why were these guidelines needed?

It has long been recognised by doctors that there is a subgroup of patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting, so called 'white coat hypertension'. If we just rely on clinic readings then such patients may be diagnosed as having hypertension when, the vast majority of the time, their blood pressure is normal.

This has led to the use of both ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM) to confirm the diagnosis of hypertension. These techniques allow a more accurate assessment of a patients' overall blood pressure. Not only does this help prevent overdiagnosis of hypertension - ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings.

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg

Diagnosing hypertension

Firstly, NICE recommend measuring blood pressure in both arms when considering a diagnosis of hypertension.

If the difference in readings between arms is more than 20 mmHg then the measurements should be repeated. If the difference remains > 20 mmHg then subsequent blood pressures should be recorded from the arm with the higher reading.

It should of course be remember that there are pathological causes of unequal blood pressure readings from the arms, such as supravalvular aortic stenosis. It is therefore prudent to listen to the heart sounds if a difference exists and further investigation if a very large difference is noted.

NICE also recommend taking a second reading during the consultation, if the first reading is > 140/90 mmHg. The lower reading of the two should determine further management.

NICE suggest offering ABPM or HBPM to any patient with a blood pressure >= 140/90 mmHg.

If the blood pressure is >= 180/120 mmHg:
- admit for specialist assessment if:
    - signs of retinal haemorrhage or papilloedema (accelerated hypertension) or
    - life-threatening symptoms such as new-onset confusion, chest pain, signs of heart failure, or acute kidney injury
- NICE also recommend referral if a phaeochromocytoma is suspected (labile or postural hypotension, headache, palpitations, pallor and diaphoresis)
- if none of the above then arrange urgent investigations for end-organ damage (e.g. bloods, urine ACR, ECG)
    - if target organ damage is identified, consider starting antihypertensive drug treatment immediately, without waiting for the results of ABPM or HBPM.
    - if no target organ damage is identified, repeat clinic blood pressure measurement within 7 days

Ambulatory blood pressure monitoring (ABPM)
- at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00)
- use the average value of at least 14 measurements

If ABPM is not tolerated or declined HBPM should be offered.

Home blood pressure monitoring (HBPM)
- for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated
- BP should be recorded twice daily, ideally in the morning and evening
- BP should be recorded for at least 4 days, ideally for 7 days
- discard the measurements taken on the first day and use the average value of all the remaining measurements

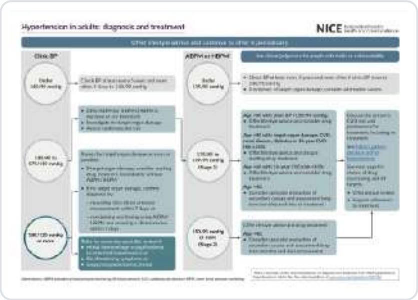
Interpreting the results

ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age



Hypertension in adults: diagnosis and treatment

NICE

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

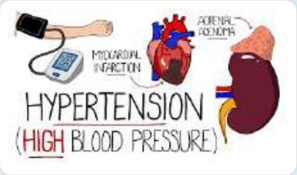
Links

NICE 👍 26 👎 37

2019 Hypertension guidelines

Suggest link Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **18.4%**

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Question 39 of 328

An 80-year-old lady is brought in by ambulance having been found unresponsive by her granddaughter.

She has a history of vascular dementia, hypertension, atrial fibrillation and mild congestive cardiac failure. She lives alone and is visited by her granddaughter, who is her main carer, once per day. Her granddaughter states she has been more confused recently, finding it hard to know the time of day, or whether she had had meals or medications, but has been otherwise well.

She takes aspirin 75mg once daily, amlodipine 5mg once daily, bisoprolol 5mg once daily, ramipril 2.5mg once daily, and simvastatin 40mg at night.

On examination in the Emergency Department she has a Glasgow coma scale of 10. Her heart rate is 30/min and her blood pressure is 68/36 mmHg. Her saturations are 95% on 60% oxygen with a respiratory rate of 30 breaths per minute.

She has a regular heart rhythm and normal first and second heart sounds with an S3 gallop. Her jugular venous pressure is raised at 6 cm and she has pitting oedema to the knees. Her capillary refill is 4 seconds and she is peripherally cool. She has fine bibasal crepitations in both lungs. Her abdomen is soft and non-tender. Her temperature is 36.1ºC.

Her ECG shows complete heart block with a ventricular rate of 30/min. Her QRS complexes are broad with a right bundle branch block pattern. There are signs of left ventricular hypertrophy but no T wave or ST segment abnormalities.

She is given 3mg of IV atropine in 500 mcg boluses but the heart rate remains 30/min with a blood pressure of 67/40 mmHg.

Which further medication is most likely to result in a sustained improvement in her heart rate?

○ Aminophylline

○ Dopamine ×

○ Glucagon ×

○ Isoprenaline ×

○ Noradrenaline ×

Submit answer

Reference ranges ⌄

Score: **18.4%**

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Question 39 of 328 ✗

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She is given 3mg of IV atropine in 500 mcg boluses but the heart rate remains 30/min with a blood pressure of 67/40 mmHg.

Which further medication is most likely to result in a sustained improvement in her heart rate?

Aminophylline 4%

Dopamine	9%
Glucagon	41%
Isoprenaline	38%
Noradrenaline	9%

This lady has severe bradycardia and is haemodynamically unstable with signs of acute heart failure.

Resus Council Advanced Life Support Guidelines advocate atropine, up to 3mg, as the initial therapy for this. However, this lady has not responded to this initial measure. The next step would be a choice of a number of drugs or transcutaneous pacing.

This lady had a history of dementia with a recent cognitive decline, lives alone and is on both a beta blocker and a calcium channel blocker. This combined with new complete heart block on her ECG, without signs of new ischaemia, is suggestive of an accidental overdose. This makes glucagon the most logical choice to further improve heart rate as it bypasses the beta blockade.

Aminophylline, isoprenaline and dopamine are all other recommended second-line drugs in unstable bradycardia, along with adrenaline (rather than noradrenaline) and could be administered if there is an unsatisfactory response to glucagon.

In practice, it is worth preparing to transcutaneously pace while furthers drugs are given, in case the bradycardia proves refractory to all medication or the clinical condition worsens further.

Reference:
Resuscitation Council (UK). Advanced Life Support. 6th Edition. London: Resuscitation Council (UK); 2011.

👍 👎 Discuss (4) Improve

Next question ›

Peri-arrest rhythms: bradycardia

The 2021 Resuscitation Council (UK) guidelines emphasise that the management of bradycardia depends on:

- 1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
- 2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

Atropine (500mcg IV) is the first line treatment in this situation.

If there is an unsatisfactory response the following interventions may be used:

- atropine, up to a maximum of 3mg
- transcutaneous pacing
- isoprenaline/adrenaline infusion titrated to response

Specialist help should be sought for consideration of transvenous pacing if there is no response to the above measures.

Resuscitation Council UK

GUIDELINES 2021

Adult bradycardia

Potential risk of asystole

The following are risk factors for asystole. Even if there is a satisfactory response to atropine specialist help is indicated to consider the need for transvenous pacing:

- complete heart block with broad complex QRS
- recent asystole
- Mobitz type II AV block
- ventricular pause > 3 seconds

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

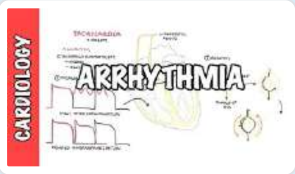
Links

Resuscitation Council UK 👍 22 👎 9

2021 Bradycardia algorithm

Suggest link Report broken link

Media



Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 0

Report broken media

Score: **17.9%**

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Question 40 of 328

A 72-year-old gentleman with previous myocardial infarctions and coronary stents presents with increased leg swelling. This has arisen over two weeks. He is still well and denies any shortness of breath but has an occasional cough at night time. He is managed on aspirin, clopidogrel, ramipril and atorvastatin.
On further questioning, he admits feeling cold at times and feels low in energy for which his GP started citalopram 2 months ago.

He denies any further chest pains. His previous records indicate disease in the circumflex and right coronary arteries previously. He admits to drinking 20-30 units of alcohol a week.

On examination, he appears well. He has bilateral pitting leg oedema with extension into the sacral area. There is a pansystolic murmur over the sternum. His abdomen is soft with a tender hepatomegaly. There is no splenomegaly, ascites, jaundice or asterixis. His JVP is raised with a double flicker pattern which peaks up to the ear lobe on the second flicker. Current blood pressure is 145/86 mmHg.

Hb	95 g/l	Na^+	137 mmol/l
Platelets	476 * 10^9/l	K^+	4.2 mmol/l
WBC	10.2 * 10^9/l	Urea	4.3 mmol/l
Neuts	9.6 * 10^9/l	Creatinine	112 µmol/l
Bilirubin	4 µmol/l	ALP	165 u/l
ALT	103 u/l	Albumin	34 g/l
PT 12.2 (14-18 normal range)	APTT 34 (34-42 normal range)		

Chest x-ray	enlarged cardiac shadow, no effusions or lung field shadowing
ECG	sinus rhythm with Q waves in V2-V3
Urine dip	2+ protein, no blood

What is the likely diagnosis?

- ○ Tricuspid regurgitation ×
- ○ Cirrhosis ×
- ○ Hypertensive cardiomyopathy ×
- ○ Nephrotic syndrome ×
- ○ Hypothyroidism ×

Submit answer

Reference ranges ∨

Score: **17.9%**

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Question 40 of 328 ×

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Bilirubin	4 µmol/l	ALP	165 u/l
ALT	103 u/l	Albumin	34 g/l
PT 12.2 (14-18 normal range)	APTT 34 (34-42 normal range)		

Chest x-ray	enlarged cardiac shadow, no effusions or lung field shadowing
ECG	sinus rhythm with Q waves in V2-V3
Urine dip	2+ protein, no blood

What is the likely diagnosis?

Tricuspid regurgitation	83%
Cirrhosis	2%
Hypertensive cardiomyopathy	6%
Nephrotic syndrome	4%
Hypothyroidism	5%

Prominent V waves on JVP → tricuspid regurgitation

Important for me Less important

This gentleman presents with leg swelling and a raised JVP which are both signs of right-sided heart failure. Nephrotic syndrome and hypothyroidism can present with swollen legs but would not have a raised JVP. Hypertensive cardiomyopathy does occur but there is no mention of previous hypertension and we do not know his blood pressure. He does not have cirrhosis given there are no signs of liver disease and his clotting is normal though his LFTs are raised. This leaves tricuspid regurgitation as the answer. He most likely developed this after suffering a posterior myocardial infarction. The gaint V waves on JVP are a classic sign.

👍 👎 Discuss (8) Improve

Next question >

Tricuspid regurgitation ★

Signs
- pan-systolic murmur
- prominent/giant V waves in JVP
- pulsatile hepatomegaly
- left parasternal heave

Causes

- right ventricular infarction
- pulmonary hypertension e.g. COPD
- rheumatic heart disease
- infective endocarditis (especially intravenous drug users)
- Ebstein's anomaly
- carcinoid syndrome

123

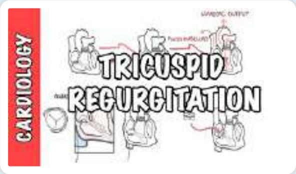
Next question

Textbooks

High-yield textbook

Extended textbook

Media


CARDIOLOGY
TRICUSPID
REGURGITATION

TyszyqQhBfc

Armando Hasudungan - YouTube 👍 1 👎 1

Tricuspid valve disease

Osmosis - YouTube 👍 1 👎 1

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Score: **17.5%**

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Question 41 of 328

A 45-year-old woman develops severe central chest pain. An ECG in the ambulance shows ST segment elevation in leads: I, aVL, V2-6. Shortly after she arrives in hospital the pain resolves and a second ECG is entirely normal. She has had three similar episodes of chest pain in the past. All episodes of chest pain have come on at rest. Blood pressure is 140/80 mmHg, heart rate is 90 beats per minute and hearts sounds are normal. She underwent coronary angiography following a previous episode of chest pain three weeks ago, which showed no significant coronary artery disease. An echocardiogram is normal.

What is the likely diagnosis?

- ○ Acute anterolateral myocardial infarction ×
- ○ Crescendo angina ×
- ○ Da Costas syndrome ×
- ○ Prinzmetal's variant angina ×
- ○ Takotsubo cardiomyopathy ×

Submit answer

Reference ranges

Score: **17.5%**

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41 -

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Question 41 of 328

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What is the likely diagnosis?

Acute anterolateral myocardial infarction	1%
Crescendo angina	3%
Da Costas syndrome	5%
Prinzmetal's variant angina	80%
Takotsubo cardiomyopathy	11%

This woman is likely to be suffering from Prinzmetal's variant angina. Classically her pain occurs at rest and the ECG demonstrates ST segment elevation that disappears as the pain abates. Normal coronary angiography supports this diagnosis, however in many Prinzmetal's angina patients there is co-existing coronary artery disease. Symptoms and ECG changes are unlikely to be reproduced with exercise testing in Prinzmetal's angina.

Prinzmetal's angina is caused by coronary artery spasm, however the underlying pathophysiology causing spasm is not currently well understood. In some patients it is associated with other vasospastic disorders such as Raynaud's phenomenon.

The main stay of treatment is:

- Avoiding precipitants of spasm such as smoking.
- Calcium channel blockers, nitrates and/or nicorandil.

Source:

http://www.escardio.org/Guidelines-&-Education/Journals-and-publications/ESC-journals-family/E-journal-of-Cardiology-Practice/Volume-11/Variant-Angina

Discuss (4) Improve

Next question

ECG: ST elevation

Causes of ST elevation

- myocardial infarction
- pericarditis/myocarditis
- normal variant - 'high take-off'
- left ventricular aneurysm
- Prinzmetal's angina (coronary artery spasm)
- Takotsubo cardiomyopathy
- rare: subarachnoid haemorrhage

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **17.1%**

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Question 42 of 328

You are reviewing in clinic a 67-year-old man who has type 2 diabetes. His glycaemic control is reasonable with metformin therapy; the latest HbA1c is 54 mmol/mol (7.1%). A few weeks ago he was noted to have a clinic blood pressure reading of 152/90 mmHg. A 24 hour blood pressure monitor was requested. The report shows his average blood pressure was 142/88 mmHg. What is the most appropriate course of action?

- ○ Do nothing for now, monitor his blood pressure regularly ×
- ○ Start an ACE inhibitor ×
- ○ Start a calcium channel blocker ×
- ○ Repeat the 24 hour blood pressure monitor in 4-8 weeks time ×
- ○ Request an ultrasound of his kidneys ×

Submit answer

Reference ranges

Score: **17.1%**

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Question 42 of 328

You are reviewing in clinic a 67-year-old man who has type 2 diabetes. His glycaemic control is reasonable with metformin therapy; the latest HbA1c is 54 mmol/mol (7.1%). A few weeks ago he was noted to have a clinic blood pressure reading of 152/90 mmHg. A 24 hour blood pressure monitor was requested. The report shows his average blood pressure was 142/88 mmHg. What is the most appropriate course of action?

Do nothing for now, monitor his blood pressure regularly	6%
Start an ACE inhibitor	80%
Start a calcium channel blocker	11%
Repeat the 24 hour blood pressure monitor in 4-8 weeks time	1%
Request an ultrasound of his kidneys	1%

Hypertension in diabetics - ACE inhibitors/A2RBs are first-line regardless of age

Important for me Less important

This patient has stage 1 hypertension as defined by NICE. He should however be treated because he has underlying diabetes.

The first-line treatment for a patient aged > 55 years is a calcium channel blocker. However, in patients with diabetes ACE inhibitors are used first-line due to their renoprotective effect.

Discuss (1) Improve

Next question ›

Diabetes mellitus: hypertension management

Patient with diabetes mellitus have traditionally had their blood pressure controlled more aggressively to help reduce their overall cardiovascular risk. However, a 2013 Cochrane review cast doubt on the wisdom of lower blood pressure targets for patients with diabetes. It compared patients who had tight blood pressure control (targets < 130/85 mmHg) with more relaxed control (< 140-160/90-100 mmHg). Patients who were more tightly controlled had a slightly reduced rate of stroke but otherwise, outcomes were not significantly different.

In light of this, NICE recommends a blood pressure target of < 140/90 mmHg for type 2 diabetics, the same as for patients without diabetes.

For patients with type 1 diabetes, NICE recommends:

> *Intervention levels for recommending blood pressure management should be 135/85 mmHg unless the adult with type 1 diabetes has albuminuria or 2 or more features of metabolic syndrome, in which case it should be 130/80 mmHg*

Because ACE inhibitors/or angiotensin-II receptor antagonist (A2RBs) have a renoprotective effect in diabetes they are the first-line antihypertensive regardless of age. A2RBs are preferred for black African or African-Caribbean diabetic patients. Further management then reverts to that of non-diabetic patients.

Remember that autonomic neuropathy may result in more postural symptoms in patients taking antihypertensive therapy.

The routine use of beta-blockers in uncomplicated hypertension should be avoided, particularly when given in combination with thiazides, as they may cause insulin resistance, impair insulin secretion and alter the autonomic response to hypoglycaemia.

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 17 👎 8

2019 Hypertension guidelines

Suggest link

Report broken link

Score: **16.7%**

1	✗
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41	✗
42	✗

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Question 43 of 328

A 54-year-old woman presents to her general practitioner complaining of a rash on her chest, face and upper arms. The rash is transient and seems to mostly occur in the summertime. She has a past medical history of hypertension, palindromic rheumatism and heart failure with preserved ejection fraction. She takes amlodipine, furosemide, indapamide and hydroxychloroquine.

On examination, there is a maculopapular rash on her chest and distal forearms. It is not itchy.

Bloods tests:

Antinuclear antibody	negative

What is the likely explanation for her symptoms?

- ○ Furosemide
- ○ Hydroxychloroquine
- ○ Indapamide
- ○ Solar urticaria
- ○ Systemic lupus erythematosus

Submit answer

Reference ranges

Score: **16.7%**

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A 54-year-old woman presents to her general practitioner complaining of a rash on her chest, face and upper arms. The rash is transient and seems to mostly occur in the summertime. She has a past medical history of hypertension, palindromic rheumatism and heart failure with preserved ejection fraction. She takes amlodipine, furosemide, indapamide and hydroxychloroquine.

On examination, there is a maculopapular rash on her chest and distal forearms. It is not itchy.

Bloods tests:

Antinuclear antibody	negative

What is the likely explanation for her symptoms?

Furosemide	8%
Hydroxychloroquine	30%
Indapamide	51%
Solar urticaria	8%
Systemic lupus erythematosus	3%

Thiazides may cause photosensitivity

Important for me Less important

Indapamide is correct. Thiazide diuretics are a well-recognised cause of a photosensitive rash. The patient's rash occurs mostly in the summertime and on sun-exposed areas and is therefore consistent with this reaction.

Furosemide is incorrect. This is mainly associated with bullous skin reactions.

Solar urticaria is incorrect. The rash, in this case, is maculopapular and non-pruritic and therefore is not consistent with a diagnosis of urticaria.

Hydroxychloroquine is incorrect. This medication is associated with rashes but is actually used to treat conditions associated with photosensitive rashes and is therefore not the likely cause.

SLE is incorrect. Photosensitive rashes are common in lupus. However, the ANA is negative, which makes a diagnosis of the systemic form of lupus, overwhelmingly unlikely.

Discuss (2) Improve

Next question ›

Thiazide diuretics ★

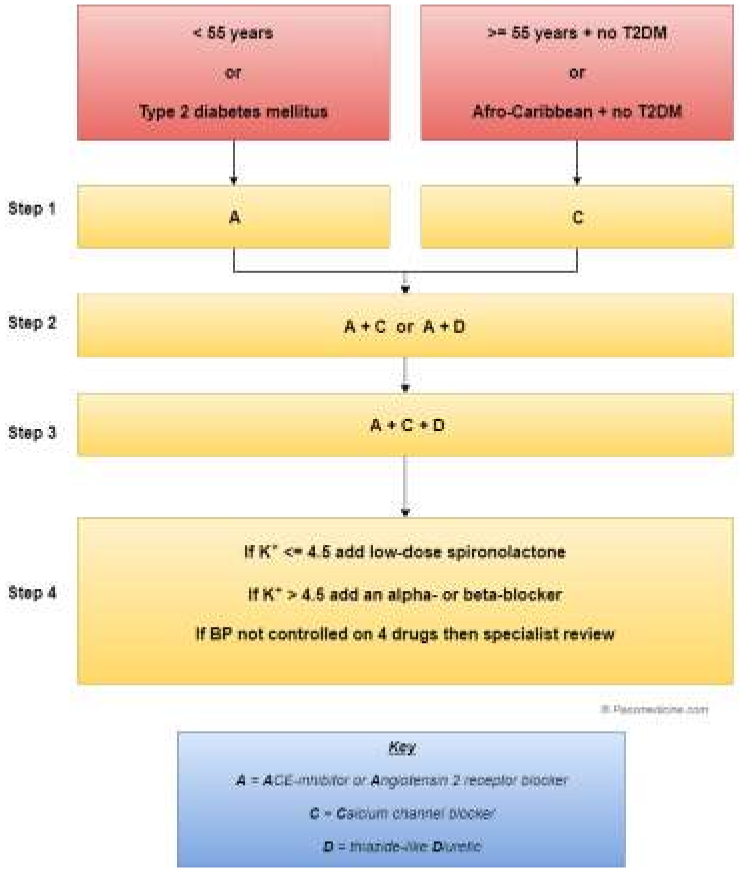
Thiazide diuretics work by inhibiting sodium reabsorption at the beginning of the distal convoluted tubule (DCT) by blocking the thiazide-sensitive Na^+-Cl^- symporter. Potassium is lost as a result of more sodium reaching the collecting ducts. Thiazide diuretics have a role in the treatment of mild heart failure although loop diuretics are better for reducing overload. The main use of bendroflumethiazide was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as indapamide and chlortalidone.

Common adverse effects
- dehydration
- postural hypotension
- hypokalaemia
    - due to increased delivery of sodium to the distal part of the distal convoluted tubule → increased sodium reabsorption in exchange for potassium and hydrogen ions
- hyponatraemia
- hypercalcaemia
    - the flip side of this is hypocalciuria, which may be useful in reducing the incidence of renal stones
- gout
- impaired glucose tolerance

- impotence

Rare adverse effects

- thrombocytopaenia
- agranulocytosis
- photosensitivity rash
- pancreatitis


< 55 years
or
Type 2 diabetes mellitus
>= 55 years + no T2DM
or
Afro-Caribbean + no T2DM
Step 1
A
C
Step 2
A + C or A + D
Step 3
A + C + D
Step 4
If K^+ <= 4.5 add low-dose spironolactone
If K^+ > 4.5 add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
© Passmedicine.com
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

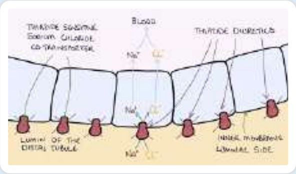
High-yield textbook

Extended textbook

Media

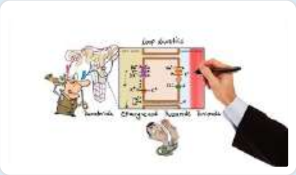
[Pharmacology - Diuretics]

Armando Hasudungan - YouTube 👍 5 👎 0



How do Thiazide Diuretics Work? Understanding Bendroflumethiazide and Indapamide

Zero To Finals - YouTube 👍 4 👎 0



Diuretics

Speed Pharmacology - YouTube 👍 2 👎 0

Report broken media

Score: **16.3%**

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Question 44 of 328

A 40-year-old man with a family history of sudden cardiac death presents to the emergency department with palpitations and exercise-induced syncope. There is no associated chest pain.

An ECG is performed and shows T wave inversion in leads V1-3.

What is the most likely underlying pathology is in this case?

○ Arrhythmogenic right ventricular cardiomyopathy ×
○ Brugada syndrome ×
○ Hypertrophic obstructive cardiomyopathy ×
○ Myocardial infarction ×
○ Pulmonary embolism ×

Submit answer

Reference ranges

Score: **16.3%**

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44 -

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A 40-year-old man with a family history of sudden cardiac death presents to the emergency department with palpitations and exercise-induced syncope. There is no associated chest pain.

An ECG is performed and shows T wave inversion in leads V1-3.

What is the most likely underlying pathology is in this case?

Arrhythmogenic right ventricular cardiomyopathy	59%
Brugada syndrome	23%
Hypertrophic obstructive cardiomyopathy	17%
Myocardial infarction	1%
Pulmonary embolism	0%

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Important for me Less important

The ECG described here is characteristic of **arrhythmogenic right ventricular cardiomyopathy**, which is consistent with the clinical presentation. This includes T wave inversion in precordial leads V1-3 (possibly also V4/5). Epsilon waves (small waves noted immediately after the QRS complex) are also seen in some cases however is not as common as T wave inversion.

Brugada syndrome is a potential hereditary cause of sudden cardiac death but is associated with ST elevation in leads V1-3 rather than the changes described here.

Hypertrophic obstructive cardiomyopathy is another potential cause of sudden cardiac death but is associated with increased precordial voltages and Q waves in the lateral, and possibly inferior, leads rather than the changes described here.

This presentation would be atypical for a **myocardial infarction**. The patient is not suffering from chest pain and instead reports symptoms that are more consistent with an arrhythmic cause. Although a myocardial infarction cannot be ruled out from this information alone, arrhythmogenic right ventricular cardiomyopathy is a more likely explanation, particularly given the strong family history.

Pulmonary emboli are associated with RV strain which can cause the T wave changes in V1-4 described in this question, however, epsilon waves are characteristic of arrhythmogenic right ventricular cardiomyopathy. Pulmonary emboli may also be associated with sinus tachycardia, right bundle branch block, right axis deviation, and S1Q3T3 patterns.

👍 👎 Discuss (3) Improve

Next question >

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology
- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation
- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

AI + Q 123

Next question >

B I

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 13 👎 7

Arrhythmogenic right ventricular cardiomyopathy ECG

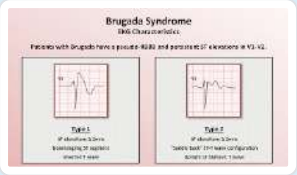
Suggest link Report broken link

Media

CARDIOLOGY
Arrhythmogenic
Right Ventricular
Cardiomyopathy
(ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome
EKG Characteristics

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

Report broken media

Score: **15.9%**

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Question 45 of 328

A 52-year-old female who you see regularly represents to your clinic after routine blood tests having recently been started on ramipril for her hypertension. Her blood pressure (BP) prior to initiation of ramipril in the clinic was 145/98mmHg and her baseline creatinine prior to treatment was 100umol/L. On review today her clinic BP appears to be well controlled at 132/84mmHg and her bloods show her creatinine has risen to 125umol/L. What is the most appropriate step in the management of her hypertension?

- ○ Reduce dose of ramipril ×
- ○ Continue current dose of ramipril ×
- ○ Stop ramipril and consider angiotensin receptor blocker ×
- ○ Increase dose of ramipril ×
- ○ Stop ramipril and consider calcium channel blocker ×

Submit answer

Reference ranges

Score: **15.9%**

1 ✗
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Question 45 of 328 ✗

A 52-year-old female who you see regularly represents to your clinic after routine blood tests having recently been started on ramipril for her hypertension. Her blood pressure (BP) prior to initiation of ramipril in the clinic was 145/98mmHg and her baseline creatinine prior to treatment was 100umol/L. On review today her clinic BP appears to be well controlled at 132/84mmHg and her bloods show her creatinine has risen to 125umol/L. What is the most appropriate step in the management of her hypertension?

Reduce dose of ramipril	2%
Continue current dose of ramipril	89%
Stop ramipril and consider angiotensin receptor blocker	3%
Increase dose of ramipril	1%
Stop ramipril and consider calcium channel blocker	5%

The main consideration here is the renal function, which shows an increase in creatinine by 25%. Her BP appears to be well controlled. The BNF recommends the angiotensin-converting enzyme inhibitors should only be stopped if the creatinine increases by 30% or eGFR falls by 25% or greater. This lady's results are within these limits and have shown good effect. It would therefore be pertinent to continue the ramipril at the current dose and monitor the renal function as per normal protocol

Discuss (2) | Improve

Next question ›

ACE inhibitors ★

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in the secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibits the conversion angiotensin I to angiotensin II
    - → decrease in angiotensin II levels → to vasodilation and reduced blood pressure
    - → decrease in angiotensin II levels → reduced stimulation for aldosterone release → decrease in sodium and water retention by the kidneys
- renoprotective mechanism
    - angiotensin II constricts the efferent glomerular arterioles
    - ACE inhibitors therefore lead to dilation of the efferent arterioles → reduced glomerular capillary pressure → decreased mechanical stress on the delicate filtration barriers of the glomeruli
    - this is particularly important in diabetic nephropathy
- ACE inhibitors are activated by phase 1 metabolism in the liver

Side-effects:

- cough
    - occurs in around 15% of patients and may occur up to a year after starting treatment
    - thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications

- pregnancy and breastfeeding - avoid
- renovascular disease - may result in renal impairment
- aortic stenosis - may result in hypotension
- hereditary of idiopathic angioedema
- specialist advice should be sought before starting ACE inhibitors in patients with a potassium >= 5.0 mmol/L

Interactions

- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day)

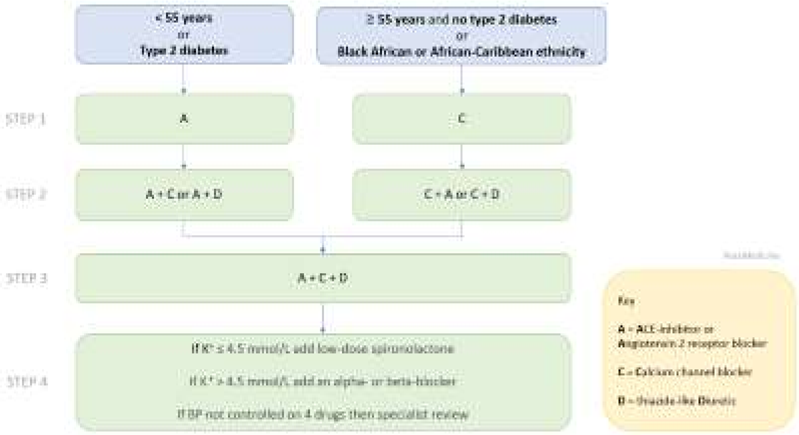
    - significantly increases the risk of hypotension

Monitoring

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
    - a rise in the creatinine and potassium may be expected after starting ACE inhibitors
    - acceptable changes are an increase in serum creatinine, up to 30% from baseline and an increase in potassium up to 5.5 mmol/l.
    - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis

Examples of common ACE inhibitors

- ramipril
- enalapril
- lisinopril



< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 53 👎 34

2014 Chronic kidney disease guidelines

BNF 👍 50 👎 25

ACE inhibitors

Suggest link Report broken link

Score: **15.6%**

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Question 46 of 328

A 43-year-old with no past medical history presents with 6 months history of exertional dyspnoea. Her echocardiogram demonstrated impaired right ventricular function with raised pulmonary arterial pressure of 78 mmHg, good systolic function and ejection fraction 80%. Right and left heart catheterisation demonstrated pulmonary arterial hypertension, with a pulmonary arterial pressure of 49/24 mmHg, V/Q scan demonstrated no chronic pulmonary emboli, CTPA no acute emboli, ultrasound abdomen no portal hypertension. She is at present comfortable at rest but is short of breath on minimal activity. What is the appropriate management?

○ No treatment ×
○ IV prostaglandin ×
○ Oral sildenafil ×
○ Atrial septostomy ×
○ List for lung transplantation ×

Submit answer

Reference ranges

Score: **15.6%**

1 ×
2 ×
3 ×
4 ×

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A 43-year-old with no past medical history presents with 6 months history of exertional dyspnoea. Her echocardiogram demonstrated impaired right ventricular function with raised pulmonary arterial pressure of 78 mmHg, good systolic function and ejection fraction 80%. Right and left heart catheterisation demonstrated pulmonary arterial hypertension, with a pulmonary arterial pressure of 49/24 mmHg, V/Q scan demonstrated no chronic pulmonary emboli, CTPA no acute emboli, ultrasound abdomen no portal hypertension. She is at present comfortable at rest but is short of breath on minimal activity. What is the appropriate management?

No treatment	6%
🟥 IV prostaglandin	20%
🟩 Oral sildenafil	63%
Atrial septostomy	2%
List for lung transplantation	10%

Treatment for pulmonary arterial hypertension is guided by the patients functional status. Patients with class I require no symptomatic treatment while those with class IV require prostaglandins, most likely intravenously. This patient is short of breath on minimal activity and hence functional class III. Endothelin receptor antagonists (e.g. bosentan) or phosphodiesterase V inhibitor (e.g. sildenafil) are appropriate. Atrial septostomy and lung transplantation are appropriate for patients who remain symptomatic at functional class IV despite intravenous prostaglandins and subsequent combination therapy with bosentan or sildenafil.

Discuss (9) | Improve

Next question >

Pulmonary arterial hypertension ★

Pulmonary arterial hypertension (PAH) may be defined as a resting mean pulmonary artery pressure of >= 20 mmHg. Endothelin is thought to play a key role in the pathogenesis of PAH. It is more common in females and typically presents between the ages of 30-50 years. Pulmonary hypertension may of course develop secondary to chronic lung diseases such as COPD - PAH is diagnosed in the absence of this although certain factors increase the risk, including HIV, cocaine and anorexigens (e.g. fenfluramine). Around 10% of cases are inherited in an autosomal dominant fashion.

Features

- progressive exertional dyspnoea is the classical presentation
- other possible features include exertional syncope, exertional chest pain and peripheral oedema
- cyanosis
- clinical signs:
    - right ventricular heave: indicating right ventricular hypertrophy or dilatation
    - loud P2: early in the disease reflects increased pulmonary artery pressure and may be accompanied by a palpable P2 in severe cases. With advanced PAH there may be right ventricular failure leading to a soft S2
    - raised JVP with prominent 'a' waves: reflects increased resistance to right atrial emptying due to elevated right ventricular end-diastolic pressure
    - tricuspid regurgitation

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that **acute vasodilator testing** is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide.

If there is a positive response to acute vasodilator testing (a minority of patients)

- oral calcium channel blockers

If there is a negative response to acute vasodilator testing (the vast majority of patients)

- prostacyclin analogues: treprostinil, iloprost
- endothelin receptor antagonists
  - non-selective: bosentan
  - selective antagonist of endothelin receptor A: ambrisentan
- phosphodiesterase inhibitors: sildenafil

Patients with progressive symptoms should be considered for a heart-lung transplant.

＋ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 12 13

2015 Guidelines for the diagnosis and treatment of pulmonary hypertension

Medcomic 14 8

Pulmonary hypertension

Respiratory Medicine journal

👍 13 👎 7

Pulmonary vasodilator testing and use of calcium channel blockers in pulmonary arterial hypertension

Suggest link

Report broken link

Media

Pulmonary hypertension

Osmosis - YouTube 👍 1 👎 4

Report broken media

Score: **15.2%**

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A 47-year-old woman presents to an endocrinology clinic with a history of hypertension. She was first diagnosed with hypertension following a private health assessment 8 years ago and at that time underwent testing to exclude any secondary causes of her hypertension. She was subsequently commenced on ramipril, which was up titrated to 10mg. When her blood pressure remained above 150/100mmHg, her GP added amlodipine 10mg and then bendroflumethiazide 2.5mg. During a recent medication review, the patient's blood pressure was noted to be 170/100mmHg. Her GP thus referred her to the endocrine team for a reconsideration of secondary hypertension. On questioning, she denies any chest pain, palpitations, change in vision, or symptoms consistent with postural hypotension. She has no family history of hypertension, does not smoke and drinks minimal alcohol.

On clinical examination, heart sounds are normal and chest clear, abdomen soft and non-tender with no organomegaly.

Her test results are as follows:

Na^+	137 mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	3.0 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
Hba1c	48 mmol/mol	(42-47)

CT chest / abdomen / pelvis shows no abnormalities.

Echocardiogram demonstrates left ventricular hypertrophy only.

Urine dip:

Blood	-
Protein	-
Glucose	Trace

Given the diagnosis, which of the following agents would be most appropriate?

- ○ Doxazosin ×
- ○ Spironolactone ×
- ○ Furosemide ×
- ○ No indication for further anti-hypertensive agents ×
- ○ Candesartan ×

Submit answer

Reference ranges ⌄

Score: **15.2%**

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Creatinine	68 µmol/L	(55 - 120)
Hba1c	48 mmol/mol	(42-47)

CT chest / abdomen / pelvis shows no abnormalities.

Echocardiogram demonstrates left ventricular hypertrophy only.

Urine dip:

Blood	-
Protein	-
Glucose	Trace

Given the diagnosis, which of the following agents would be most appropriate?

Doxazosin	62%
Spironolactone	27%
Furosemide	3%
No indication for further anti-hypertensive agents	2%
Candesartan	6%

Poorly controlled hypertension, already taking an ACE inhibitor, calcium channel blocker and a standard-dose thiazide diuretic. K+ > 4.5mmol/l - add an alpha- or beta-blocker

Important for me Less important

This patient has resistant hypertension, that is to say, hypertension which has not responded to three appropriately dosed anti-hypertensive agents. Effective treatment is needed given her evidence of end-organ damage (left ventricular hypertrophy).

Given that her potassium is > 4.5, spironolactone would not be recommended.

Furosemide would not be recommended in a patient without evidence of heart failure prior to the trial of alpha blockade.

Candesartan should not be co-prescribed with ramipril.

Doxazosin (an alpha-blocker) or alternatively a beta-blocker, would be the most appropriate 4th line antihypertensive agent.

👍 👎 Discuss (3) Improve

Next question >

Hypertension: management ★

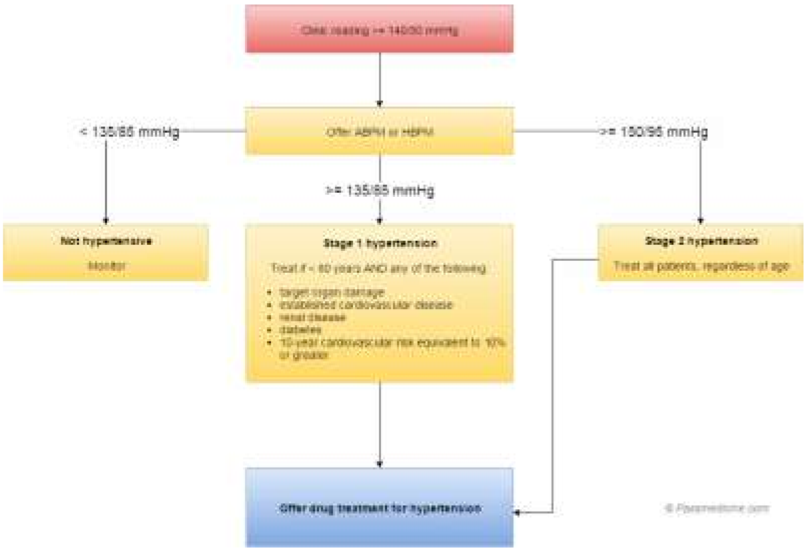
NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 135/85 mmHg
>= 150/95 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

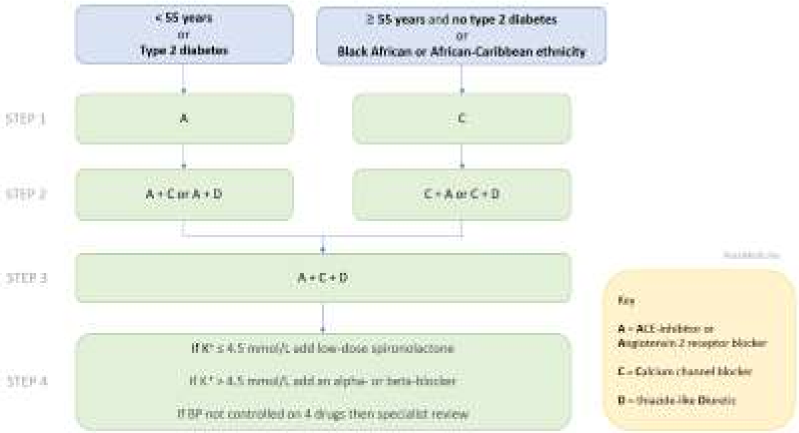
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.



< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
  - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

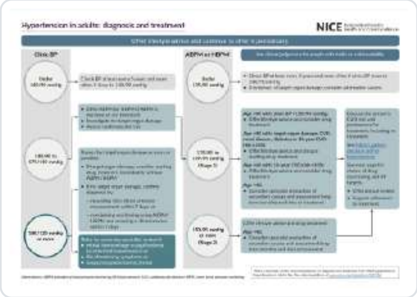
- add a third drug to make, i.e.:
  - if already taking an **(A + C)** then add a **D**
  - if already **(A + D)** then add a **C**
- **(A + C + D)**

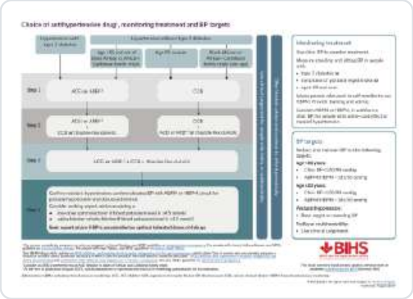
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
    - confirm elevated clinic BP with ABPM or HBPM
    - assess for postural hypotension.
    - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*


Hypertension in adults: diagnosis and treatment
NICE


Choice of antihypertensive drug, monitoring treatment and BP targets
BIHS

Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg

	Clinic BP	ABPM / HBPM
Age > 80 years	150/90 mmHg	145/85 mmHg

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

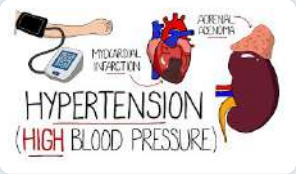
Links

NICE 👍 162 👎 162

2019 Hypertension guidelines

Suggest link Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **17%**

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47	✓

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A 64-year-old male presents sudden onset back pain while painting a wall at home, radiating to his left anterior chest and jaw, associated with nausea and vomiting. It appears to be constant for the past 3 hours since onset and is his first ever episode. His past medical history includes hypertension, hypercholesterolaemia and one previous transient ischaemic attack.

His ECG demonstrates left ventricular hypertrophy by voltage criteria and T-wave inversion in I, aVF and V5 and V6. On examination, his heart sounds are both present with a soft systolic murmur. The chest is clear and abdomen is soft and non-tender without a pulsatile mass. Both radial pulses are intact with no delay. There is a mild radio-femoral delay. His blood pressure is stable at 134/80 mmHg, heart rate 124/min and sinus rhythm. His bloods are as follows:

Hb	123 g/l
MCV	82 fl
Platelets	204 * 10^9/l
WBC	9.2 * 10^9/l
Troponin T	240 (normal range < 32)

His pain settles transiently with 2.5mg of subcutaneous morphine. What is the most appropriate immediate action?

○ Aspirin 300mg and clopidogrel 300mg and treatment dose low molecular weight heparin ×

○ Intravenous fluids ×

○ Blood transfusion ×

○ Coronary angiogram +/- stent as appropriate ×

○ CT aorta ×

Submit answer

Reference ranges

Score: **17%**

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Question 48 of 328 ✓

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MCV	82 fl
Platelets	204 * 10^9/l
WBC	9.2 * 10^9/l
Troponin T	240 (normal range < 32)

His pain settles transiently with 2.5mg of subcutaneous morphine. What is the most appropriate immediate action?

Aspirin 300mg and clopidogrel 300mg and treatment dose low molecular weight heparin	22%
Intravenous fluids	1%
Blood transfusion	0%
Coronary angiogram +/- stent as appropriate	15%
CT aorta	62%

The combination of an atypical history of back and chest pain, persistent pain, non-specific ECG changes, mildly raised troponin and an unexplained sinus tachycardia should raise suspicion that this is not a simple diagnosis of acute coronary syndrome. The clue lies in the distal pulses: the lack of radial-radial delay suggests any aortic pathology occurs below the subclavian artery origin. The mild radial-femoral delay suggests a descending artery lesion. The patient is unwell but not actively exsanguinating as evidenced by the stable Hb and blood pressure. An underlying diagnosis of acute aortic syndrome, a new term classifying all acute aortic pathology including dissections, ulcers and aneurysms, must be ruled out by high definition CT aorta with contrast.

👍 👎 Discuss (4) Improve

Next question >

Aortic dissection: features and classification ★

Aortic dissection is a rare but serious cause of chest pain.

Pathophysiology
- tear in the tunica intima of the wall of the aorta

Associations
- hypertension: the most important risk factor
- trauma
- bicuspid aortic valve
- collagens: Marfan's syndrome, Ehlers-Danlos syndrome
- Turner's and Noonan's syndrome
- pregnancy
- syphilis

Features:
- chest/back pain
  - typically severe and 'sharp', 'tearing' in nature
  - pain is typically maximal at onset

    - classically chest pain is more common in type A dissection and upper back pain is more common in type B dissection. However, there is considerable overlap and both chest and back pain are present in many patients
- pulse deficit
    - weak or absent carotid, brachial, or femoral pulse
    - variation (>20 mmHg) in systolic blood pressure between the arms
- aortic regurgitation
- hypertension
- other features may result from the involvement of specific arteries. For example:
    - coronary arteries → angina
    - spinal arteries → paraplegia
    - distal aorta → limb ischaemia
- the majority of patients have no or non-specific ECG changes. In a minority of patients, ST-segment elevation may be seen in the inferior leads

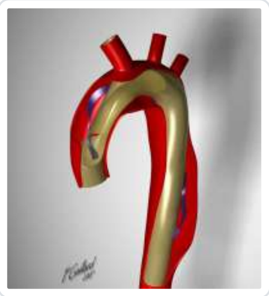
Classification

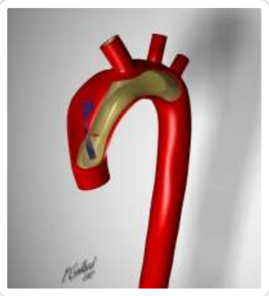
Stanford classification

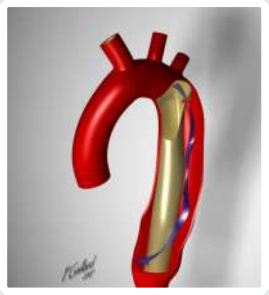
- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally







123

Next question >

Textbooks

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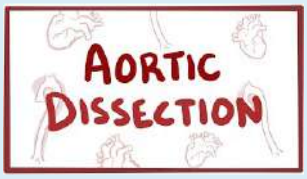
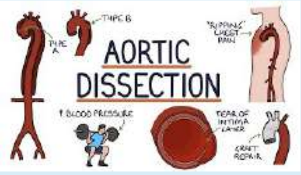
Media


AORTIC
DISSECTION

Aortic dissection

Osmosis - YouTube 👍 6 👎 0


AORTIC
DISSECTION
TYPE B
TYPE A
"RIPPING" CHEST PAIN
↑ BLOOD PRESSURE
TEAR OF INTIMA LAYER
GRAFT REPAIR

Understanding Aortic Dissection

Zero To Finals - YouTube 👍 1 👎 1

Report broken media

Score: **18.8%**

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48	✓

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Question 49 of 328

A 54-year-old man is reviewed on the cardiac ward some 2 days after admission with an acute coronary syndrome event. He has suffered an inferior myocardial infarction some 4 years ago, for which he was stented, and takes aspirin together with atorvastatin, ramipril, bisoprolol and indapamide. Examination on the ward is unremarkable, and he is deemed fit for discharge.

Which of the following is the most appropriate option for anti-platelet therapy on discharge?

- ○ Aspirin and clopidogrel ×
- ○ Aspirin only ×
- ○ Aspirin and ticagrelor ×
- ○ Aspirin and dipyridamole ×
- ○ Aspirin and prasugrel ×

Submit answer

Reference ranges

Score: **18.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

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Which of the following is the most appropriate option for anti-platelet therapy on discharge?

Aspirin and clopidogrel	20%
Aspirin only	20%
Aspirin and ticagrelor	40%
Aspirin and dipyridamole	0%
Aspirin and prasugrel	20%

Nice guidelines now recommend secondary prevention with ticagrelor and aspirin, not clopidogrel and aspirin after ACS

Important for me Less important

NICE guidance is clear for patients who are medically managed for acute coronary syndrome, it states that:

Prescribe DAPT (dual anti-platelet therapy). This is aspirin 75 mg daily plus ticagrelor 90 mg twice a day for 12 months. Ticagrelor is the preferred choice (even in those previously treated with clopidogrel) unless the bleeding risk outweighs the benefit. For people at high risk of bleeding, continue DAPT for at least one month.
For people with MI at high ischaemic risk who have tolerated DAPT without a bleeding complication, consider treatment with DAPT (ticagrelor 60 mg twice daily) in addition to aspirin for longer than 12 months and up to 36 months. If ticagrelor is not suitable, consider

clopidogrel 75 mg daily (as well as aspirin) for longer than 12 months.

Aspirin and prasugrel is only recommended for patients undergoing percutaneous intervention and stenting, and aspirin and dipyridamole was formerly used for secondary prevention of stroke. This has now been replaced for secondary prevention of stroke by clopidogrel alone.

👍 👎 Discuss (12) Improve

Next question >

Adenosine diphosphate (ADP) receptor inhibitors

★

Examples include:

- Clopidogrel
- Prasugrel
- Ticagrelor
- Ticlopidine

Mechanism of ADP receptor inhibitors

- Adenosine diphosphate (ADP) is one of the main platelet activation factors, mediated by G-coupled receptors P2Y1 and P2Y12.
- The main target of ADP receptor inhibition is the P2Y12 receptor, as it is the one which leads to sustained platelet aggregation and stabilisation of the platelet plaque.

Evidence

- As aspirin and ADP inhibitors work by blocking different platelet aggregation pathways, their potential synergy has been studied by multiple clinical trials, particularly in high-risk patients presenting with acute coronary syndrome (ACS) or undergoing percutaneous coronary intervention (PCI).
- Clopidogrel used to be the most commonly used ADP inhibitor, however, due to its interindividual variability in antiplatelet effects, newer agents such as prasugrel and ticagrelor have been developed.
- Multiple trials since have shown a marked reduction in short- and long-term ischaemic events when using prasugrel and aspirin,

compared to clopidogrel and aspirin in moderate- to high-risk ACS patients.[1]

- Current NICE guidelines for ACS, therefore, recommend starting dual antiplatelet treatment (DAPT) with Aspirin (75mg daily) and Ticagrelor (90mg twice daily) for 12 months, as a secondary prevention strategy.
- NICE guidelines for people with ACS undergoing PCI recommend aspirin (75-100mg daily) in combination with either prasugrel (10mg daily), ticagrelor (90mg twice daily), or clopidogrel (75mg daily, if prasugrel or ticagrelor are not suitable) for 12 months, with aspirin alone thereafter.[2]

Notable adverse effects

- ticagrelor may cause dyspnoea
  - due to the impaired clearance of adenosine

Interactions and contraindications

- A drug interaction exists between clopidogrel and proton pump inhibitors, particularly omeprazole and esomeprazole, leading to reducing antiplatelet effects.
- Patients with prior stroke or transient ischaemic attack, high risk of bleeding, and prasugrel hypersensitivity are absolute contraindications to prasugrel use.
- Ticagrelor is contraindicated in patients with a high risk of bleeding, those with a history of intracranial haemorrhage, and those with severe hepatic dysfunction. It is also to be used with caution in those with acute asthma or COPD, as ticagrelor-treated patients experience higher rates of dyspnoea.[1]

+ Q 123

Next question ›

Textbooks

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Extended textbook

Media

Thrombolytics

Anticoagulants and antiplatelets

Speed Pharmacology - YouTube 👍 3 👎 0

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Question 50 of 328

A 58 year-old man with a background of is ischaemic heart disease and Crohn's disease has developed colonic enterocutaneous fistulae. He is admitted to hospital under the surgical team and a temporary ileostomy is formed to defunction the bowel and promote healing.

Two days post-operatively he develops palpitations and the surgical team request your assistance.

On examination the pulse rate is 220bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on 2L nasal oxygen.

The chest is clear to auscultation.

A 12-lead ECG reveals a wide-complex tachycardia with a polymorphic waveform.

Blood tests from the morning reveal:

Hb	129 g/l
Platelets	643 * 10^9/l
WBC	13.8 * 10^9/l
Na^+	129 mmol/l
K^+	3.3 mmol/l
Phosphate	0.63 mmol/l
Mg^{++}	0.59 mmol/l
Urea	8.1 mmol/l
Creatinine	97 µmol/l
Bilirubin	15 µmol/l
ALP	143 u/l
ALT	53 u/l
Albumin	31 g/l

What is the most appropriate initial management?

○ Adenosine 6mg IV ×

○ Magnesium sulphate 2g ×

○ Metoprolol 5mg IV ×

○ Synchronised DC shock ×

○ Amiodarone 300mg IV ×

Submit answer

Reference ranges

Score: **20.4%**

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Urea	8.1 mmol/l
Creatinine	97 µmol/l
Bilirubin	15 µmol/l
ALP	143 u/l
ALT	53 u/l
Albumin	31 g/l

What is the most appropriate initial management?

Adenosine 6mg IV	2%
Magnesium sulphate 2g	86%
Metoprolol 5mg IV	1%
Synchronised DC shock	3%
Amiodarone 300mg IV	9%

In this scenario this patient has developed polymorphic ventricular tachycardia with no adverse features. This is likely to be secondary to electrolyte derangement in this case.

Initial management steps include stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate 2 g IV over 10 min (= 8 mmol).

If adverse features are present (shock, syncope, myocardial ischaemia or heart failure) arrange immediate synchronised cardioversion.

👍 👎 Discuss (6) Improve

Next question ›

Ventricular tachycardia: management ★

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure, syncope) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks.

Drug therapy

- amiodarone: ideally administered through a central line

- lidocaine: use with caution in severe left ventricular impairment
- procainamide

Verapamil should NOT be used in VT.

If drug therapy fails
- electrophysiological study (EPS)
- implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

+ Q 123

Next question >

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Links

Resuscitation Council UK 👍 43 👎 7

2021 Tachycardia algorithm

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Question 51 of 328

A 83 year old lady presents to heart failure follow up clinic. She has a history of NSTEMI and gallstones. Her symptoms are reasonably poorly controlled. She is able to walk around her bungalow, but struggles to walk to her local shops 100m away due to breathlessness.

She had recently seen her GP who added spironolactone to her regular medications due to persistent hypokalaemia. Her potassium has since normalised.

Her latest echo reveals an ejection fraction of 25%. Her ECG is sinus rhythm Her last BNP was 1000 pg/ml.

She is currently taking senna, ramipril 10mg, aspirin 75m, frusemide 40mg bd, simvastatin 40mg, and spironolactone 50mg.

Her observations at clinic are:
- oxygen saturations: 94% on room air
- blood pressure: 126/66 mmHg
- heart rate: 84/min

Which additional medication would be beneficial?

○ Ivabradine

○ Bisoprolol

○ Digoxin

○ Diltiazem

○ Atenolol

Submit answer

Reference ranges ⌵

Score: **20%**

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Her observations at clinic are:
- oxygen saturations: 94% on room air
- blood pressure: 126/66 mmHg
- heart rate: 84/min

Which additional medication would be beneficial?

Ivabradine	29%
Bisoprolol	65%
Digoxin	2%
Diltiazem	1%
Atenolol	2%

This patient is already taking some prognostically beneficial medications; ACE inhibitor and aldosterone antagonist. The additional of a beta blocker would be a beneficial medication; both from a preventative of re-modelling and a reduction in heart rate.

Heart rate is a well established modifiable risk factor, which when appropriately controlled can improve morbidity and mortality in heart failure. This patient's heart rate is not well controlled.

Bisoprolol, carvedilol, nebivolol and metoprolol are the only evidence-based cardioselective beta blockers for heart failure patients.

Ivabradine is an If (funny) channel blocker, which reduces heart rate by blocking the If current in the sinoatrial node. NICE guidelines advocate the use of ivabradine for heart failure in a select group of patients:

- Ejection fraction <35%
- Heart rate >75/min
- Sinus rhythm
- NYHA class 2-4
- Maximally titrated beta blocker therapy.

Since this patient is not on maximal beta blocker therapy, it would not be appropriate to commence ivabradine. Diltiazem may be used as an anti-anginal medication, but this patient does not have angina. Digoxin may be of benefit in heart rate, especially if the patient has atrial fibrillation, but should only be considered following failure of first and second line therapies.

👍 👎 Discuss (5) Improve

Next question >

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a

beta-blocker

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

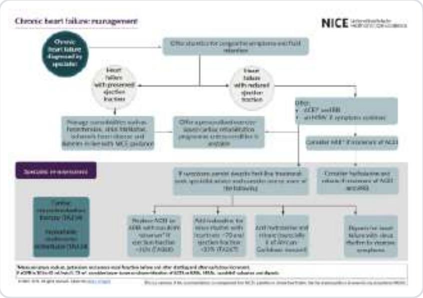
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs

    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years



123

Next question

Textbooks

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Links

NICE 👍 14 👎 12

2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

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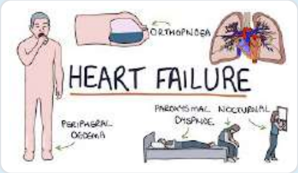
Media


ORTHOPNOEA
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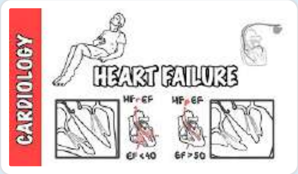
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
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Heart failure

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A 72-year-old man attends the cardiology clinic. He has a past medical history of heart failure. He advises you that he has had worsening shortness of breath over the last few weeks. He is now symptomatic at rest. His regular medications include ramipril, bisoprolol, furosemide and eplerenone. His observations are as follows: heart rate 70 beats per minute, blood pressure 125/75 mmHg, respiratory rate 22 breaths per minute, and SpO2 94% on air. On examination, you note pitting oedema to the mid calves and bilateral coarse crepitations.

An echocardiogram and ECG is performed:

Echo	Left ventricular failure with an ejection fraction of 25%

ECG	Sinus rhythm; QRS 140 msec; Dominant S wave in V1 and a broad monophasic R wave in lateral leads (I, aVL, V5-6)

What treatment is most likely to be beneficial to long-term survival?

- ○ Bendroflumethiazide ×
- ○ Bumetanide ×
- ○ Cardiac resynchronisation therapy with defibrillator (CRT-D) ×
- ○ Cardiac resynchronisation therapy with pacing (CRT- P) ×
- ○ Ivabradine ×

Submit answer

Reference ranges

Score: **19.6%**

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Question 52 of 328 ✗

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An echocardiogram and ECG is performed:

Echo	Left ventricular failure with an ejection fraction of 25%

ECG	Sinus rhythm; QRS 140 msec; Dominant S wave in V1 and a broad monophasic R wave in lateral leads (I, aVL, V5-6)

What treatment is most likely to be beneficial to long-term survival?

Bendroflumethiazide	2%
Bumetanide	1%
Cardiac resynchronisation therapy with defibrillator (CRT-D)	48%
Cardiac resynchronisation therapy with pacing (CRT- P)	40%
Ivabradine	8%

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

Important for me Less important

The patient has symptomatic (NYHA class IV) cardiac failure requiring escalation of treatment. He is already on an ACE-I, beta blocker, mineralocorticoid receptor antagonist, and loop diuretic. He has severe left ventricular systolic dysfunction (LVSD) with an ejection fraction of 25%. It is important to note the presence of LBBB with a significantly prolonged QRS. The dominant S wave in V1 and a broad monophasic R wave in the lateral leads are classic of LBBB.

Cardiac resynchronisation therapy with pacing (CRT- P) is correct. Cardiac resynchronisation therapy (CRT) is recommended as a treatment option for people with heart failure who have left ventricular dysfunction with a left ventricular ejection fraction (LVEF) of 35% or less, and LBBB with a QRS duration > 130 msec. The patient in this clinical case, therefore, fulfils these criteria. Many randomised controlled trials have shown that CRT significantly prolongs survival in patients with heart failure. The NICE guidelines have criteria on whether to choose CRT-D or CRT-P. In patients with NYHA class IV (as seen in this case), CRT-P is the preferred modality. In this case, it would also be important to consider the use of sacubitril/valsartan. The PROVE-HF trial (as well as case reports and observational studies) have shown that sacubitril/valsartan is able to induce reverse cardiac remodeling, which has the potential to avoid ICD or CRT implantation.

Bendroflumethiazide is incorrect. Bendroflumethiazide can be used for mild or moderate heart failure. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

Bumetanide is incorrect. Bumetanide is a type of loop diuretic which has been shown to have greater bioavailability compared to furosemide due to increased absorption in patients who have oedematous bowels. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

Cardiac resynchronisation therapy with defibrillator (CRT-D) is incorrect. As the patient has NYHA class IV symptomatology, CRT-P is the correct option as per NICE guidelines.

Ivabradine is incorrect. Ivabradine is recommended as an option for treating chronic heart failure for patients who are in sinus rhythm with a heart rate of 75 beats per minute or more and who are already on standard therapy with ACE inhibitors, beta-blockers and aldosterone antagonists. The patient in this clinical case has a heart rate of 70 beats per minute and ivabradine is therefore not indicated.

👍 👎 Discuss (5) Improve

Next question >

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy
- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training
- improves symptoms but not hospitalisation/mortality

+ Q 123

Next question >

Textbooks

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Links

NICE 👍 14 👎 4

Cardiac resynchronisation therapy for the treatment of heart failure

NICE 👍 5 👎 1

2018 Chronic heart failure guidelines

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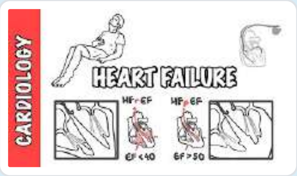
Media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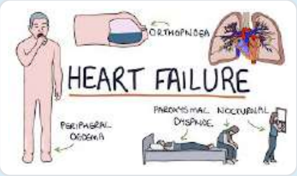
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51	✗
52	✗

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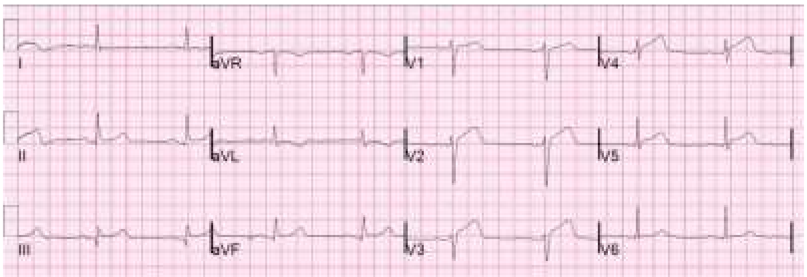
A 79-year-old man presents with severe central chest pain which started around 90 minutes ago. He is known to have ischaemic heart disease and had a coronary artery bypass graft (CABG) five years ago. On arrival in the Emergency Department he is clammy and vomiting. An ECG is taken:



© Image used on license from Dr Smith, University of Minnesota

What is the most accurate description of what is shown on this ECG?

(MI = myocardial infarction, STEMI = ST elevation myocardial infarction, NSTEMI = non-ST elevation myocardial infarction)

○ Normal ECG

○ Current inferior NSTEMI

○ Current anterior NSTEMI

○ Current inferior STEMI

○ Current anterior STEMI

Submit answer

Reference ranges

Score: **19.2%**

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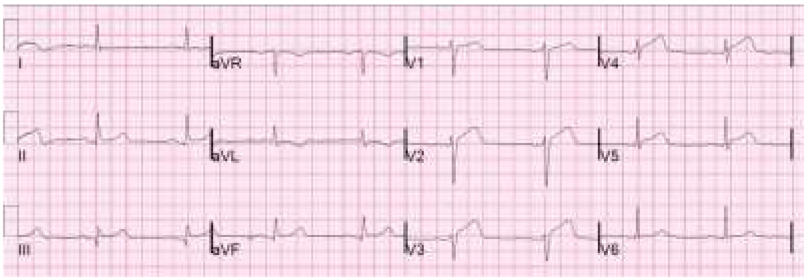
Question 53 of 328

A 79-year-old man presents with severe central chest pain which started around 90 minutes ago. He is known to have ischaemic heart disease and had a coronary artery bypass graft (CABG) five years ago. On arrival in the Emergency Department he is clammy and vomiting. An ECG is taken:



© Image used on license from Dr Smith, University of Minnesota

What is the most accurate description of what is shown on this ECG?

(MI = myocardial infarction, STEMI = ST elevation myocardial infarction, NSTEMI = non-ST elevation myocardial infarction)

Normal ECG	3%
Current inferior NSTEMI	3%
Current anterior NSTEMI	7%
Current inferior STEMI	25%
Current anterior STEMI	61%

Ischaemic changes in leads V1-V4 - left anterior descending

Important for me Less important

In the anterior leads (V2-4) ST elevation can clearly be seen indicating a STEMI. Angiogram revealed an acutely occluded saphenous vein graft to the left anterior descending which was opened with percutaneous coronary intervention.

In the inferior leads there is a pathological Q wave in lead III and non-diagnostic Q waves in leads II and aVF (see below for definitions). These changes suggest that this patient has previously had an inferior MI.

The older and simpler definition of a pathological Q wave:

- Q-wave of >=0.04 s and an amplitude >=25% of the R-wave in that lead

The new (and much more complicated) Joint European Society of Cardiology/American College of Cardiology definition of a pathologic Q wave is:

- any Q-wave in leads V2 - V3 >= 0.02 s or QS complex in leads V2 and V3
- Q-wave >= 0.03 s and > 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4 - V6 in any two leads of a contiguous lead grouping (I, aVL,V6; V4 - V6; II, III, and aVF)
- R-wave >= 0.04 s in V1 - V2 and R/S >= 1 with a concordant positive T-wave in the absence of a conduction defect

👍 👎 Discuss (9) Improve

Next question ›

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary

	ECG changes	**Coronary artery**
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

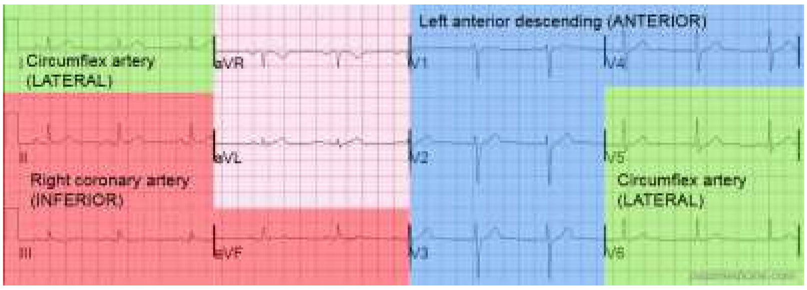

Circumflex artery (LATERAL)
aVR
Left anterior descending (ANTERIOR)
V1
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question >

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal ECG correlation with coronary anatomy	👍 24	👎 40
University of Massachusetts Medical School Classic circumflex infarction ECG	👍 14	👎 23

Suggest link

Report broken link

Score: **18.9%**

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Question 54 of 328

A 45-year-old woman presents to the emergency department after a road traffic accident where she sustains multiple injuries including an open fracture of her left tibia and fibula. The following day she has an open reduction and internal fixation of the left tibia and fibula and remains in hospital for physiotherapy. She is quite immobile during this period and then develops subsequent painful swelling and erythema of the left calf. Subsequent ultrasonography confirms a left-sided above knee deep vein thrombosis.

Before treatment starts, she develops sudden onset weakness in her right leg and right arm, dysarthric speech and a reduction in conscious level. Subsequent CT scanning confirms the presence of a left-sided infarct in the middle cerebral artery territory. Doppler investigation of the carotids shows a 20% stenosis on the left side and 10% on the right side. The 24-hour tape shows average heart rate 52bpm with 1.5s pauses maximum, sinus bradycardia

Which of the following features from further investigations would best explain this woman's presentation?

- ○ Anti phospholipid antibody positive ×
- ○ Protein C deficiency ×
- ○ Patent foramen ovale (PFO) ×
- ○ Hypercholesterolaemia ×
- ○ Dilated cardiomyopathy on ECHO ×

Submit answer

Reference ranges

35	✗
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A 45-year-old woman presents to the emergency department after a road traffic accident where she sustains multiple injuries including an open fracture of her left tibia and fibula. The following day she has an open reduction and internal fixation of the left tibia and fibula and remains in hospital for physiotherapy. She is quite immobile during this period and then develops subsequent painful swelling and erythema of the left calf. Subsequent ultrasonography confirms a left-sided above knee deep vein thrombosis.

Before treatment starts, she develops sudden onset weakness in her right leg and right arm, dysarthric speech and a reduction in conscious level. Subsequent CT scanning confirms the presence of a left-sided infarct in the middle cerebral artery territory. Doppler investigation of the carotids shows a 20% stenosis on the left side and 10% on the right side. The 24-hour tape shows average heart rate 52bpm with 1.5s pauses maximum, sinus bradycardia

Which of the following features from further investigations would best explain this woman's presentation?

Anti phospholipid antibody positive	4%
Protein C deficiency	3%
Patent foramen ovale (PFO)	90%
Hypercholesterolaemia	1%
Dilated cardiomyopathy on ECHO	1%

This unfortunate lady has developed a thrombus as a result of her recent operation and subsequent immobility. Part of the thrombus has embolised into her left middle cerebral artery.

One would normally expect the source of the embolus to be from the carotids or from the heart. However, the minimal carotid stenosis and lack of history of atrial fibrillation make this less likely. The finding of a

PFO on an ECHO would explain how a thrombus from the leg would reach the brain rather than the lungs.

PFOs are extremely common and have been found in 25-30% of people at autopsy. PFOs have been linked to an increased risk of stroke. There is also some evidence that subclinical DVTs in the presence of a PFO may be one cause for cryptogenic stroke http://content.onlinejacc.org/article.aspx?articleid=1120253

The evidence is however limited that PFO closure will reduce the risk of stroke.

The other options except E are all risk factors for stroke but cannot explain the mechanism of embolism with near-normal carotids and sinus rhythm. E is not associated with an increased risk of embolic stroke.

👍 👎 Discuss (5) Improve

Next question >

Patent foramen ovale ★

Patent foramen ovale (PFO) is present in around 20% of the population. It may allow embolus (e.g. from DVT) to pass from right side of the heart to the left side leading to a stroke - 'a paradoxical embolus'

There also appears to be an association between migraine and PFO. Some studies have reported improvement in migraine symptoms following closure of the PFO.

The management of patients with PFO who've had a stroke remains controversial. Options include antiplatelet therapy, anticoagulant therapy or PFO closure.

AI + Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Score: **18.5%**

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A 52-year-old man of Afro-Caribbean descent is reviewed after having ambulatory blood pressure monitoring (ABPM). This had been ordered after a works medical recorded a blood pressure of 156/86 mmHg. He smokes 20 cigarettes/day and has a family history of ischaemic heart disease. The results show the following:

Average daytime blood pressure: 142/88 mmHg
QRISK 10 year cardiovascular risk: 23%
Urine dipstick: NAD
ECG: sinus rhythm, rate 78/min

Na^+	141 mmol/l
K^+	4.6 mmol/l
Urea	4.5 mmol/l
Creatinine	82 µmol/l
Total cholesterol	5.4 mmol/l
HDL cholesterol	0.9 mmol/l
Fasting glucose	5.3 mmol/l

What is the most appropriate course of action?

○ Start treatment with a calcium channel blocker

○ Diagnose stage 1 hypertension and advise about lifestyle changes

○ Start treatment with an ACE inhibitor

○ Start treatment with a thiazide-like diuretic

○ Repeat the ABPM

Submit answer

31	✓
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38	✗
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Question 55 of 328 ✗

A 52-year-old man of Afro-Caribbean descent is reviewed after having ambulatory blood pressure monitoring (ABPM). This had been ordered after a works medical recorded a blood pressure of 156/86 mmHg. He smokes 20 cigarettes/day and has a family history of ischaemic heart disease. The results show the following:

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QRISK 10 year cardiovascular risk: 23%
Urine dipstick: NAD
ECG: sinus rhythm, rate 78/min

Na^+	141 mmol/l
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Urea	4.5 mmol/l
Creatinine	82 µmol/l
Total cholesterol	5.4 mmol/l
HDL cholesterol	0.9 mmol/l
Fasting glucose	5.3 mmol/l

What is the most appropriate course of action?

Start treatment with a calcium channel blocker	70%
Diagnose stage 1 hypertension and advise about lifestyle changes	18%
Start treatment with an ACE inhibitor	10%
Start treatment with a thiazide-like diuretic	1%
Repeat the ABPM	1%

Stage 1 hypertension is defined by an ABPM reading of >= 135/85 mmHg, with stage 2 hypertension having a cut-off of >= 150/95 mmHg.

This patient therefore has stage 1 hypertension. We should therefore treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 20% or greater, as is present in this case.

ACE inhibitors are usually the treatment of choice for patients < 55-years-old but as this patient is of Afro-Caribbean descent a calcium channel blocker should be used instead.

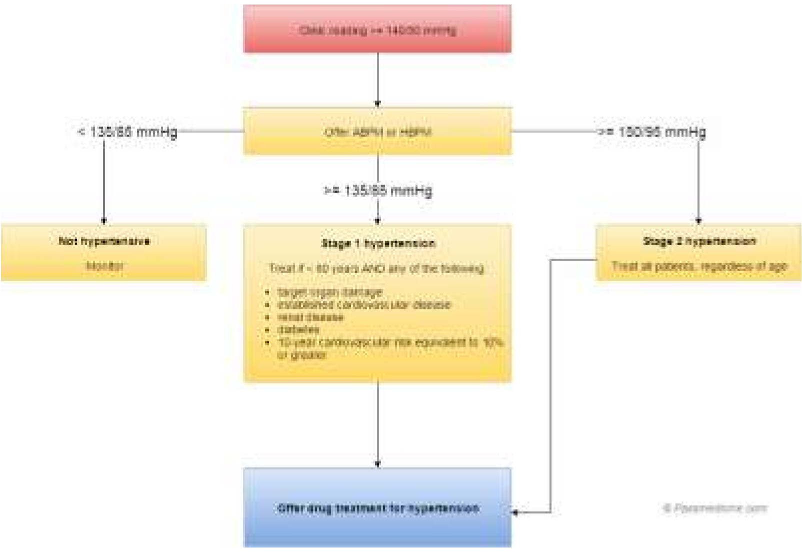
Discuss (6) | Improve

Next question >

Hypertension: diagnosis ★

NICE published updated guidelines for the management of hypertension in 2019. This builds on the significant guidelines released in 2011 that recommended:

- classifying hypertension into stages
- recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
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Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Why were these guidelines needed?

It has long been recognised by doctors that there is a subgroup of patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting, so called 'white coat hypertension'. If we just rely on clinic readings then such patients may be diagnosed as having hypertension when, the vast majority of the time, their blood pressure is normal.

This has led to the use of both ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM) to confirm the diagnosis of hypertension. These techniques allow a more accurate assessment of a patients' overall blood pressure. Not only does this help prevent overdiagnosis of hypertension - ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings.

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	**Criteria**
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg

Diagnosing hypertension

Firstly, NICE recommend measuring blood pressure in both arms when considering a diagnosis of hypertension.

If the difference in readings between arms is more than 20 mmHg then the measurements should be repeated. If the difference remains > 20 mmHg then subsequent blood pressures should be recorded from the arm with the higher reading.

It should of course be remember that there are pathological causes of unequal blood pressure readings from the arms, such as supravalvular aortic stenosis. It is therefore prudent to listen to the heart sounds if a difference exists and further investigation if a very large difference is noted.

NICE also recommend taking a second reading during the consultation, if the first reading is > 140/90 mmHg. The lower reading of the two should determine further management.

NICE suggest offering ABPM or HBPM to any patient with a blood pressure >= 140/90 mmHg.

If the blood pressure is >= 180/120 mmHg:

- admit for specialist assessment if:
  - signs of retinal haemorrhage or papilloedema (accelerated hypertension) or
  - life-threatening symptoms such as new-onset confusion, chest pain, signs of heart failure, or acute kidney injury
- NICE also recommend referral if a phaeochromocytoma is suspected (labile or postural hypotension, headache, palpitations, pallor and diaphoresis)
- if none of the above then arrange urgent investigations for end-organ damage (e.g. bloods, urine ACR, ECG)
  - if target organ damage is identified, consider starting antihypertensive drug treatment immediately, without waiting for the results of ABPM or HBPM.
  - if no target organ damage is identified, repeat clinic blood pressure measurement within 7 days

Ambulatory blood pressure monitoring (ABPM)

- at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00)
- use the average value of at least 14 measurements

If ABPM is not tolerated or declined HBPM should be offered.

Home blood pressure monitoring (HBPM)

- for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated
- BP should be recorded twice daily, ideally in the morning and evening
- BP should be recorded for at least 4 days, ideally for 7 days
- discard the measurements taken on the first day and use the average value of all the remaining measurements

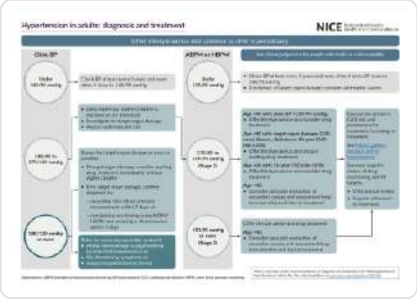
Interpreting the results

ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age



123

Next question

Textbooks

High-yield textbook

Extended textbook

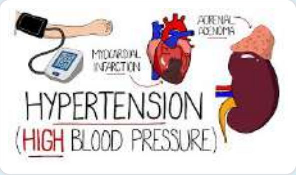
Links

NICE 👍 26 👎 37

2019 Hypertension guidelines

Suggest link Report broken link

Media


MYOCARDIAL INFARCTION
ADRENAL ADENOMA
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **18.2%**

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Question 56 of 328

A patient with a severe rheumatic heart disease is about to attend a gastroscopy with oesophageal dilatation. He has no current active gastrointestinal infection. What endocarditis prophylaxis is recommended?

- ○ Ciprofloxacin PO 30 min pre-procedure ×
- ○ None ×
- ○ IV ampicillin at time of surgery ×
- ○ IV cefazolin at time of surgery ×
- ○ Erythromycin PO 30 min pre-procedure ×

Submit answer

Reference ranges

Score: **18.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
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7 ✗
8 ✗

9	✗
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Question 56 of 328

A patient with a severe rheumatic heart disease is about to attend a gastroscopy with oesophageal dilatation. He has no current active gastrointestinal infection. What endocarditis prophylaxis is recommended?

Ciprofloxacin PO 30 min pre-procedure	3%
None	87%
IV ampicillin at time of surgery	6%
IV cefazolin at time of surgery	3%
Erythromycin PO 30 min pre-procedure	2%

Prophylaxis is only indicated when one of the following are met:

Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
Previous infective endocarditis
Cardiac transplantation with the subsequent development of cardiac valvulopathy
Congenital heart disease but only if it involves: Unrepaired cyanotic defects, including palliative shunts and conduits Completely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure (after which the prosthetic material is likely to have been endothelialised) Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation

Congenital heart disease but only if it involves:

- Unrepaired cyanotic defects, including palliative shunts and conduits
- Completely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure (after which the prosthetic material is likely to have been endothelialised)
- Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation)

Active or not active gastrointestinal infection does not alter your management of prophylaxis, but would be advisable to defer the procedure if possible.

👍 👎 Discuss (1) Improve

Next question >

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
    - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
    - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis
    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*

    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link Report broken link

Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **17.9%**

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A 56-year-old gentleman was admitted to the Emergency Department with a three-hour history of severe central chest pain. The pain came on suddenly whilst he was walking. The pain has remained severe in intensity since onset. He also felt short of breath at rest with associated clamminess. There was no history of trauma. His past medical history comprised of hypertension, hypercholesterolaemia and gout. He was prescribed amlodipine 5mg OD, ramipril 7.5mg OD, atorvastatin 20mg OD, allopurinol 100mg and orlistat 120mg TDS. He smoked 20 cigarettes per day and consumed 30 units of alcohol per week.

On examination, he appeared pale and clammy. His blood pressure was 196/78 mmHg in his right arm and 152/65 mmHg in his left arm. His heart rate was 92bpm. His oxygen saturations were 96% on air. Examination of his cardiovascular system revealed the presence of a prominent early diastolic murmur in the aortic area. Examination of his respiratory system revealed tachypnoea but no added sounds on auscultation. Examination of his gastrointestinal and neurological systems was unremarkable.

He was admitted to the resuscitation bay and commenced on 15 litres of oxygen per minute. He was administered IV diamorphine 2.5mg OD. Investigations conducted revealed the following results:

ECG: normal sinus rhythm 96 bpm, ST elevation in leads II, III and aVF
Chest x-ray: normal appearances of heart and lung

What is the best immediate management step?

- ○ Commence thrombolysis
- ○ Transfer for immediate percutaneous coronary intervention
- ○ Commence aspirin 300mg OD and clopidogrel 300mg OD
- ○ Conduct myocardial perfusion scan
- ○ Commence IV labetalol infusion

Submit answer

Reference ranges

Score: **17.9%**

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ECG: normal sinus rhythm 96 bpm, ST elevation in leads II, III and aVF
Chest x-ray: normal appearances of heart and lung

What is the best immediate management step?

Commence thrombolysis	3%
Transfer for immediate percutaneous coronary intervention	30%
Commence aspirin 300mg OD and clopidogrel 300mg OD	8%
Conduct myocardial perfusion scan	1%
Commence IV labetalol infusion	58%

This patient has had an aortic dissection involving the ascending aorta and arch of the aorta, as manifested by the presence of severe chest pain, aortic regurgitation, unequal blood pressures and evidence of an inferior myocardial infarction on ECG. In this situation, thrombolysis is absolutely contraindicated as the myocardial infarction is due to dissection of the vessels and not due to an atherosclerotic thrombotic occlusion. Options 2 and 3 are therefore also not suitable. This gentleman requires urgent intravenous beta blockade for aggressive blood pressure control and to act as a negative inotrope. He will also require an urgent cardiothoracic surgical opinion and further imaging to confirm the diagnosis.

👍 👎 Discuss (9) Improve

Next question >

Aortic dissection: investigation and management

★

Classification

Stanford classification

- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally

Investigation

It's important to remember that patients may present acutely and be clinically unstable. The choice of investigations will have to take account of this.

Chest x-ray
- widened mediastinum

CT angiography of the chest, abdomen and pelvis is the investigation of choice
- suitable for stable patients and for planning surgery
- a false lumen is a key finding in diagnosing aortic dissection

Transoesophageal echocardiography (TOE)
- more suitable for unstable patients who are too risky to take to CT scanner

Management

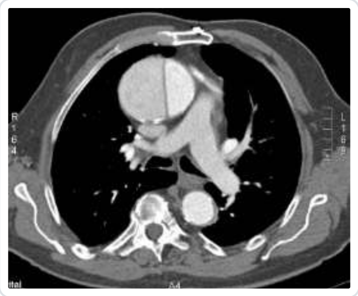
Type A
- surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention

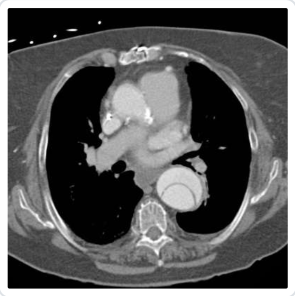
Type B*
- conservative management
- bed rest
- reduce blood pressure IV labetalol to prevent progression

Complications

Complications of backward tear
- aortic incompetence/regurgitation
- MI: inferior pattern is often seen due to right coronary involvement

Complications of a forward tear
- unequal arm pulses and BP
- stroke
- renal failure





*endovascular repair of type B aortic dissection may have a role in the future

123

Next question

Textbooks

High-yield textbook

Extended textbook

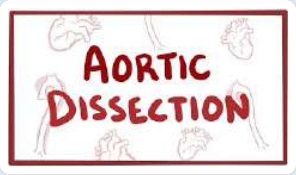
Links

American Heart Association 13 13

2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease

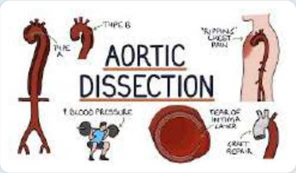
Suggest link Report broken link

Media


AORTIC DISSECTION

Aortic dissection

Osmosis - YouTube 👍 9 👎 0


TYPE B
TYPE A
AORTIC DISSECTION
"RIPPING" CHEST PAIN
↑ BLOOD PRESSURE
TEAR OF INTIMA LAYER
GRAFT REPAIR

Understanding Aortic Dissection

Zero To Finals - YouTube 👍 1 👎 0

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Score: **19.3%**

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A 75-year-old female is admitted to hospital with community-acquired pneumonia. She is currently being treated with intravenous benzylpenicillin and oral clarithromycin. On the third day of admission, she complains of a sensation of fullness on the lateral aspect of her right leg.

Her past medical history includes osteoarthritis, temporal arteritis (now off steroids) and a uterine prolapse.

Bloods performed the day before show:

Hb	131 g/l
Platelets	$520 * 10^9$/l
WBC	$14.2 * 10^9$/l

Na^+	141 mmol/l
K^+	4.1 mmol/l
Urea	6.2 mmol/l
Creatinine	86 µmol/l
CRP	78 mg/L

A doppler ultrasound scan is performed that shows a superficial vein thrombosis in the right leg. What is the most appropriate management?

○ Treatment dose low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for three months

○ Prophylactic dose low molecular weight heparin (LMWH) for 30 days.

○ Treatment dose low molecular weight heparin (LMWH) for 30 days.

○ Non-steroidal anti-inflammatory drugs for 8-12 days. ×

○ Low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for six months. ×

Submit answer

Reference ranges ∨

Score: **19.3%**

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Treatment dose low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for three months 41%

Prophylactic dose low molecular weight heparin (LMWH) for 30 days. 11%

Treatment dose low molecular weight heparin (LMWH) for 30 days. 13%

Non-steroidal anti-inflammatory drugs for 8-12 days. 22%

Low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for six months. 14%

Superficial vein thrombosis (also referred to as thrombophlebitis) is a common condition and in the majority of cases is self-limiting. Patients presenting with a superficial vein thrombosis are at increased risk of a venous thromboembolic event and up to 20% of patients will already have a deep vein thrombosis (DVT) on presentation. Patients are more likely to have thrombus extension into the deep venous system if they have more than 5cm of thrombus within 10cm of the saphenofemoral junction (i.e. within the proximal long saphenous vein).

The evidence for how superficial vein thrombosis should be managed comes from a Cochrane review and is used to support the Scottish intercollegiate guidelines network (SIGN) guidelines on the prevention and management of venous thromboembolism (guide 122). The guidelines recommend that all patients with clinical signs of a superficial vein thrombosis should have an ultrasound scan to exclude a DVT.

Patients with a confirmed diagnosis of a superficial vein thrombosis should be considered for treatment with anti-embolism stockings and prophylactic doses of low molecular weight heparin (LMWH) for 30 days or fondaparinux for 45 days. In cases where LMWH is contraindicated, 8-12 days of oral non-steroidal anti-inflammatory drugs should be offered as this has been shown to reduce the risk of thrombus extension.

The full guideline can be found at:
http://www.sign.ac.uk/pdf/sign122.pdf

👍 👎 Discuss (11) Improve

Next question >

Superficial thrombophlebitis ★

Superficial thrombophlebitis, as the name suggests describes the inflammation associated with thrombosis of one of the superficial veins, usually the long saphenous vein of the leg. This process is usually non-infective in nature but secondary bacterial infection may rarely occur resulting in septic thrombophlebitis.

Around 20% with superficial thrombophlebitis will have an underlying deep vein thrombosis (DVT) at presentation and 3-4% of patients will progress to a DVT if untreated. The risk of DVT is partly linked to the length of vein affected - an inflammed vein > 5 cm is more likely to have an associated DVT.

Management

There are currently a variety of treatment approaches to superficial thrombophlebitis. Traditionally NSAIDs have been used, with topical NSAIDs for limited and mild disease and oral NSAIDs for more severe disease.

Topical heparinoids have also be used in the management of superficial thrombophlebitis.

A Cochrane review however found topical NSAIDs and heparinoids have no significant benefit in terms of reducing extension or progression to DVT. Oral NSAIDs were however shown to reduce the risk of extension by 67%.

Compression stockings are also used. Remember that the ankle-brachial pressure index (ABPI) should be measured before prescribing compression stockings, particularly if using class 2 or above stockings.

One of the major changes to the management of superficial thrombophlebitis is the increased use of low-molecular weight heparin. This has been shown to reduce extension and transformation to DVT. SIGN produced guidelines in 2010:

> *Patients with clinical signs of superficial thrombophlebitis affecting the proximal long saphenous vein should have an ultrasound scan to exclude concurrent DVT.*
>
> - *Patients with superficial thrombophlebitis should have anti-embolism stockings and can be considered for treatment with prophylactic doses of LMWH for up to 30 days or fondaparinux for 45 days.*

> - *If LMWH is contraindicated, 8-12 days of oral NSAIDS should be offered.*
>
> *Patients with superficial thrombophlebitis at, or extending towards, the sapheno-femoral junction can be considered for therapeutic anticoagulation for 6-12 weeks.*

This may be a significant departure from our current practice - the majority of patients with superficial thrombophlebitis (i.e. those affecting the long saphenous vein) should be referred for an ultrasound scan.

AI + Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries Superficial thrombophlebitis	👍 6 👎 2

Suggest link Report broken link

Media

Thrombophlebitis

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **19%**

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A 76-year-old man presents to the emergency department with complaints of shortness of breath on exertion, difficulty walking up stairs, and significant weight gain over the last 2 weeks. His daughter who is with him expresses concerns about him and says that he is needing to lay upright in his armchair in order to get any sleep.

His medical history includes 2 previous non-ST-segment elevation myocardial infarctions for which he was treated with coronary stenting and hypertension. Physical examination shows a heart rate of 111 beats per minute and blood pressure of 113/76 mmHg. He is visibly short of breath at rest and is using accessory muscles. His JVP is 7cm above the angle of Louis, his apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of his chest reveals audible crepitations bi-basally and he has 2+ peripheral oedema extending to his mid-thighs. An ECG shows Q-waves in his inferior leads, and his bloods show elevated troponins.

Which of the following is the most appropriate next step in the management of this patient?

- ○ Obtain an urgent echocardiogram
- ○ Prescribe oral furosemide and discharge from the emergency department with a view to follow up with his GP
- ○ Load him with aspirin and clopidogrel
- ○ Commence bisoprolol to control his heart rate
- ○ Administer a bolus of intravenous furosemide and then start him on an infusion

Submit answer

Reference ranges

Score: **19%**

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Which of the following is the most appropriate next step in the management of this patient?

Obtain an urgent echocardiogram	23%
Prescribe oral furosemide and discharge from the emergency department with a view to follow up with his GP	2%
Load him with aspirin and clopidogrel	7%
Commence bisoprolol to control his heart rate	2%
Administer a bolus of intravenous furosemide and then start him on an infusion	65%

The most appropriate medication in patients with acute heart failure and a preserved ejection fraction who have signs of volume overload is addition/up-titration of a loop diuretic

Important for me   Less important

This patient presents with acute decompensated congestive heart failure. He has evidence of significant volume overload on exam, with an elevated JVP and signs of pulmonary and peripheral oedema. The aetiology of his heart failure is likely to be ischaemic cardiomyopathy related to his history of myocardial infarctions and manifests with a displaced apical impulse and a holosystolic murmur heard loudest at the apical region, suggestive of significant ventricular dilatation. His symptoms are severe and he requires inpatient hospitalisation and treatment with intravenous loop diuretics to improve his symptoms and to optimise his volume status.

An echocardiogram would be useful in the evaluation of this patient but is not the most appropriate next step as he is currently significantly symptomatic and needs intravenous loop diuretics to improve his volume status.

Although this patient does need to be treated with loop diuretics, he is in significant distress and likely needs to be admitted as an inpatient and should be treated more aggressively with intravenous loop diuretics.

This patient does have a history of ischaemic heart disease and is at risk of having further ischemic events. However, the findings on his ECG represent an old inferior infarct, and there a no features to suggest an acute event. In addition, he may have elevated troponins simply as a result of having ischaemic cardiomyopathy. He does not necessarily need to be loaded on dual antiplatelet therapy at this moment, but he should have his troponins trended to ensure there is no significant rise in their values.

Starting a beta-blocker in acute decompensated heart failure is contraindicated.

👍 👎 Discuss (6) Improve

Next question >

Heart failure (acute): features ★

Acute heart failure (AHF) is a life-threatening emergency. AHF is a term used to describe the sudden onset or worsening of the symptoms of heart failure. Thus it may present with or without a background history of pre-existing heart failure. AHF without a past history of heart failure is called de-novo AHF. Decompensated AHF is more common (66-75%) and presents with a background history of HF.

It usually presents after the age of 65 years and is a major cause of unplanned hospital admission in such patients.

AHF is usually caused by a reduced cardiac output that results from a functional or structural abnormality.

De-novo heart failure is caused by increased cardiac filling pressures and myocardial dysfunction usually as a result of ischaemia. This causes reduced cardiac output and therefore hypoperfusion. This, in turn, can cause pulmonary oedema. Other less common causes of de-novo AHF are:

- Viral myopathy
- Toxins
- Valve dysfunction

Decompensated heart failure accounts for most cases of AHF. The most common precipitating causes of acute AHF are:

- Acute coronary syndrome
- Hypertensive crisis
- Acute arrhythmia
- Valvular disease

There is generally a history of pre-existing cardiomyopathy. It usually presents with signs of fluid congestion, weight gain, orthopnoea and breathlessness.

Patients with heart failure are broadly characterised into 1 of 4 groups based on whether they present:

- With or without hypoperfusion
- With or without fluid congestion

Classifying patients into one of these 4 groups is clinically useful as it determines the therapeutic approach.

Generally speaking, the signs and symptoms of AHF are as follows:

Symptoms	Signs
Breathlessness	Cyanosis
Reduced exercise tolerance	Tachycardia
Oedema	Elevated jugular venous pressure
Fatigue	Displaced apex beat
	Chest signs: classically bibasal crackles but may also cause a wheeze
	S3-heart sound

Sometimes the presentation will be that of the underlying cause (e.g: chest pain, viral infection)

Over 90% of patients with AHF have a normal or increased blood pressure (mmHg).

The diagnostic workup for patients with AHF includes:

- Blood tests
  - this is to look for any underlying abnormality such as anaemia, abnormal electrolytes or infection.
- Chest X-ray
  - findings include pulmonary venous congestion, interstitial oedema and cardiomegaly
- Echocardiogram
  - recommended for patients with new-onset heart failure and for patients with known heart failure who have a suspected change in cardiac function
  - particularly important if cardiogenic shock or suspected valvular problems
  - may also identify post-myocardial infarction complications
- B-type natriuretic peptide
  - raised levels (>100mg/litre) indicate myocardial damage and are supportive of the diagnosis.

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **20.3%**

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Question 60 of 328

An 89-year-old gentleman presented to the general medical clinic with complaints of palpitations. He was diagnosed with atrial fibrillation (AF) three months ago and started on bisoprolol. This was part of a plan to rate control, rather than rhythm control, his AF. The bisoprolol has been up-titrated as his symptoms did not come under control with his dose. Palpitations occur almost every day but are not associated with any other symptoms. He also has a history of ischaemic heart disease with a myocardial infarction five years ago and type 2 diabetes mellitus. He currently takes bisoprolol, metformin, aspirin and simvastatin. He has declined anticoagulation with warfarin or NOAC despite the risk as his wife died from an intracranial bleed whilst taking warfarin.

On examination, his heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema.How should he be further managed?

- ○ Amiodarone
- ○ Dronedarone
- ○ Amlodipine
- ○ Diltiazem
- ○ Left atrial ablation

Submit answer

Reference ranges

Score: **20.3%**

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An 89-year-old gentleman presented to the general medical clinic with complaints of palpitations. He was diagnosed with atrial fibrillation (AF) three months ago and started on bisoprolol. This was part of a plan to rate control, rather than rhythm control, his AF. The bisoprolol has been up-titrated as his symptoms did not come under control with his dose. Palpitations occur almost every day but are not associated with any other symptoms. He also has a history of ischaemic heart disease with a myocardial infarction five years ago and type 2 diabetes mellitus. He currently takes bisoprolol, metformin, aspirin and simvastatin. He has declined anticoagulation with warfarin or NOAC despite the risk as his wife died from an intracranial bleed whilst taking warfarin.

On examination, his heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema.How should he be further managed?

Amiodarone	35%
Dronedarone	6%
Amlodipine	9%
Diltiazem	21%
Left atrial ablation	29%

The correct answer is diltiazem. This is a gentleman with symptomatic, but not decompensating, permanent AF which is not responding to first-line treatment with a beta-blocker (bisoprolol). NICE advises that first line treatment for a rate-control strategy in AF should be either a beta-blocker or a rate-limiting calcium channel blocker, but digoxin can be considered for non-paroxysmal AF in patients who are not very active. If the first-line treatment fails, either with continuing symptoms or poor response of ventricular rate then a combination therapy with any of the following two can be used: beta-blocker, diltiazem, digoxin. Amiodarone, dronedarone and left atrial ablation are all strategies for cardioversion. Amlodipine is a calcium channel blocker but is used in hypertension rather than in AF as it is not rate-limiting, and would therefore not help.

Source:
'Atrial Fibrillation: Management.' The National Institute for Health and Care Excellence, June 2014.

Discuss (15) | Improve

Next question

Atrial fibrillation: classification

An attempt was made in the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines to simplify and clarify the classification of atrial fibrillation (AF).

It is recommended that AF be classified into 3 patterns:

- first detected episode (irrespective of whether it is symptomatic or self-terminating)
- recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term **paroxysmal AF** is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term **persistent AF** is used. Such episodes usually last greater than 7 days
- in **permanent AF** there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 3

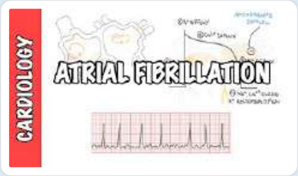
2021 Atrial fibrillation guidelines

Suggest link Report broken link

Media

Atrial fibrillation

Osmosis - YouTube 👍 2 👎 0


CARDIOLOGY
ATRIAL FIBRILLATION

Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 1 👎 1

Report broken media

Score: **20%**

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Question 61 of 328

A 65-year-old Caucasian man attends the dermatology clinic with a 3-week history of a new rash. He complains of a discolouration of his forearms and hands that initially appeared whilst on holiday in the south of France. It is mildly itchy but not painful. There is no history of a change in diet or detergents and he has no known allergies. His past medical history includes hypertension, type 2 diabetes, ischaemic heart disease and atrial fibrillation.

On examination, there is a purplish discolouration of his hands up to his elbows bilaterally. His face and scalp are mildly erythematous. There is no blistering or crusting.

Which of the following is most likely to be responsible for this patient's presentation?

- ○ Aspirin
- ○ Digoxin
- ○ Indapamide
- ○ Metformin
- ○ Ramipril

Submit answer

Reference ranges

Score: **20%**

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Question 61 of 328 ✗

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On examination, there is a purplish discolouration of his hands up to his elbows bilaterally. His face and scalp are mildly erythematous. There is no blistering or crusting.

Which of the following is most likely to be responsible for this patient's presentation?

Aspirin	13%
Digoxin	21%
Indapamide	57%
Metformin	1%
Ramipril	8%

Thiazides may cause photosensitivity

Important for me Less important

This patient has drug-induced photosensitivity, a skin reaction secondary to UV radiation causing expected burns or dermatitis in patients taking photosensitising medications. Drugs which cause phototoxicity include: antibiotics (tetracyclines, sulfonamides, fluoroquinolones), NSAIDs, diuretics, sulfonylureas, antipsychotics as well as amiodarone, quinine and hydroxychloroquine. The clinical features of photosensitivity can vary according to the medication taken as well as the type of reaction. Some reactions may be phototoxic whilst others are photoallergic. A phototoxic reaction results from direct damage mediated by UV activation of the photosensitising agent. These reactions can appear within minutes to hours after exposure and appears as a sunburn reaction with reddening and swelling. Rarely, the skin may change pigmentation (e.g. blue-green discoloration seen with amiodarone). In severe reactions, blisters and vesicles may be seen. Other symptoms may include itch, as seen in this scenario. Thiazides typically cause a phototoxic reaction. In contrast, a photoallergic reaction is a cell-mediated response. These are less common and present as an eczematous, itchy skin reaction within 24-72 hours after exposure to sunlight. Unlike phototoxic reactions, photoallergic reactions are capable of spreading to areas that have not been exposed to sunlight. The treatment of drug-induced photosensitivity is to avoid the trigger, if possible. If the medication is essential to be taken then protective measures should be taken including sunscreen or protective clothing.

In toxic doses, digoxin is capable of causing side effects including arrhythmias, dizziness, yellow or green visual changes and skin reactions. However, they do not commonly cause photosensitivity. If there are any concerns regarding digoxin toxicity, a digoxin level can be taken with the therapeutic window ranging between 0.7ng/mL and 2.0ng/mL.

Aspirin is a common cause of acute or chronic urticaria. However, it does not cause photosensitivity.

Metformin and ramipril do not commonly cause a rash or skin discolouration. In rare cases, patients may have an allergic reaction to these medications which results in the eruption of urticaria and, in extreme cases, lip and tongue swelling and airway compromise. However, they do not cause photosensitivity.

👍 | 👎 | Discuss (3) | Improve

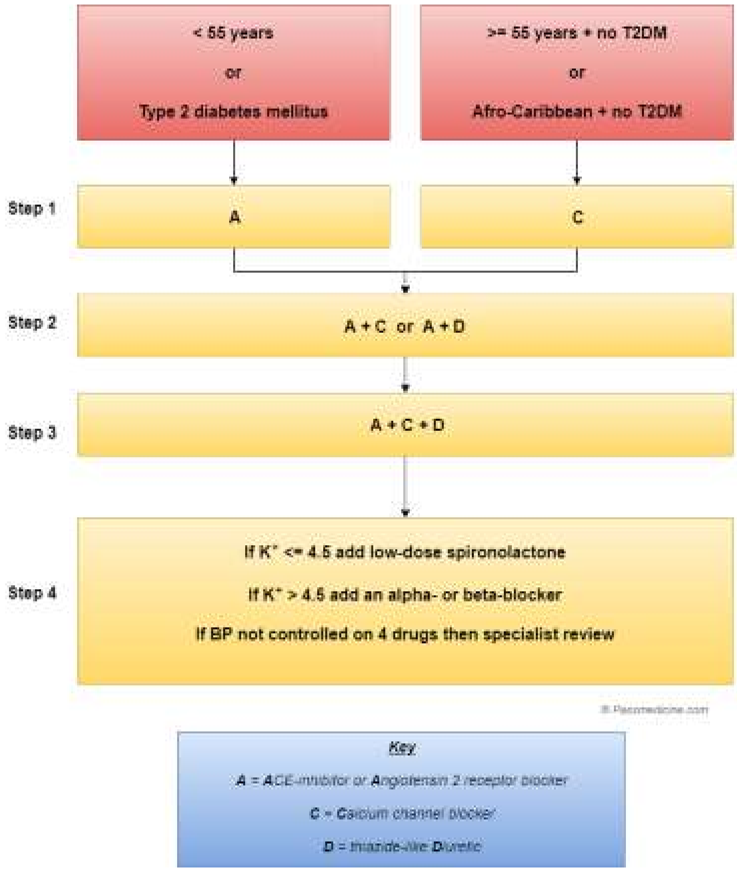
Next question >

Thiazide diuretics ★

Thiazide diuretics work by inhibiting sodium reabsorption at the beginning of the distal convoluted tubule (DCT) by blocking the thiazide-sensitive Na^{+}-Cl^{-} symporter. Potassium is lost as a result of more sodium reaching the collecting ducts. Thiazide diuretics have a role in the treatment of mild heart failure although loop diuretics are better for reducing overload. The main use of bendroflumethiazide was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as indapamide and chlortalidone.

Common adverse effects
- dehydration
- postural hypotension
- hypokalaemia
    - due to increased delivery of sodium to the distal part of the distal convoluted tubule → increased sodium reabsorption in exchange for potassium and hydrogen ions
- hyponatraemia
- hypercalcaemia
    - the flip side of this is hypocalciuria, which may be useful in reducing the incidence of renal stones
- gout
- impaired glucose tolerance
- impotence

Rare adverse effects
- thrombocytopaenia
- agranulocytosis
- photosensitivity rash
- pancreatitis


< 55 years
or
Type 2 diabetes mellitus
>= 55 years + no T2DM
or
Afro-Caribbean + no T2DM
Step 1
A
C
Step 2
A + C or A + D
Step 3
A + C + D
Step 4
If K⁺ <= 4.5 add low-dose spironolactone
If K⁺ > 4.5 add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
© Passmedicine.com
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

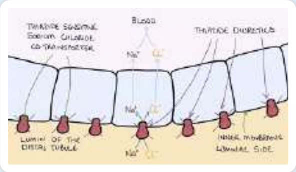
High-yield textbook

Extended textbook

Media

Pharmacology - Diuretics

Armando Hasudungan - YouTube 👍 5 👎 0



How do Thiazide Diuretics Work? Understanding Bendroflumethiazide and Indapamide

Zero To Finals - YouTube 👍 4 👎 0

Diuretics

Speed Pharmacology - YouTube 👍 2 👎 0

Report broken media

Score: **19.7%**

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Question 62 of 328

An 89-year-old lady presents acutely short of breath and distressed. She has a background of previous myocardial infarction (MI) and hypertension. She is now coughing up white sputum. Examination reveals bilateral course crepitations throughout the lung fields. She has a raised jugular venous pressure and peripheral oedema. Observations are a respiratory rate of 35/min, oxygen saturations of 92% on 15 litres of oxygen per minute, blood pressure 135/90 mmHg, heart rate 100/min. Chest X-ray reveals widespread interstitial shadowing. Intravenous furosemide has been given but the patient fails to improve. Which of the following would be useful in treating this patient?

- ○ Intravenous antibiotics ×
- ○ Oral furosemide ×
- ○ Bilevel positive airway pressure ×
- ○ Continuous positive airway pressure ×
- ○ Chest physiotherapy ×

Submit answer

Reference ranges

Score: **19.7%**

1 ×
2 ×
3 ×
4 ×

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Question 62 of 328 ✗

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Intravenous antibiotics	1%
Oral furosemide	1%
Bilevel positive airway pressure	12%
Continuous positive airway pressure	85%
Chest physiotherapy	1%

Acute heart failure not responding to treatment - consider CPAP

Important for me Less important

The patient is suffering from severe pulmonary oedema with bilateral course crackles and cough productive of white sputum. The patient has signs of right sided heart failure with raised JVP and peripheral oedema. They also have a history of MI and hypertension that are two risk factors for heart failure.

NICE guidance on Acute Heart Failure 2014 states that a patient who has failed medical management of pulmonary oedema with severe dyspnoea should be considered for CPAP. BIPAP is not used in acute pulmonary oedema.

👍 | 👎 | 💬 Discuss (5) | Improve

Next question >

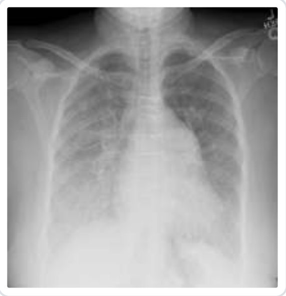
Heart failure: acute management ★

Recommended treatments for all patients

- IV loop diuretics
  - e.g. furosemide or bumetanide

Possible additional treatments

- oxygen
  - this should be given in line with British Thoracic Society guidelines, i.e. keep oxygen saturations at 94-98%
- vasodilators
  - nitrates should not be routinely given to all patients
  - they may, however, have a role if concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease
  - the major side-effect/contraindication is hypotension



Patients with respiratory failure

- CPAP

Patients with hypotension (e.g. < 85 mmHg)/cardiogenic shock

- this can be a difficult scenario to manage
  - some of the treatments typically used for acute heart failure (e.g. loop diuretics and nitrates) may exacerbate the hypotension
- inotropic agents
  - e.g. dobutamine

    - should be considered for patients with severe left ventricular dysfunction who have potentially reversible cardiogenic shock
- vasopressor agents
    - e.g. norepinephrine
    - normally only used if insufficient response to inotropes and evidence of end-organ hypoperfusion
- mechanical circulatory assistance
    - e.g. intra-aortic balloon counterpulsation or ventricular assist devices

Other treatments
- opiates
    - NICE state 'do not routinely offer opiates to people with acute heart failure'
    - they were previously used routinely and help to reduce dyspnoea/distress in patients
    - however some studies have suggested increased morbidity in patients given opiates

General points
- regular medication for heart failure such as beta-blockers ACE-inhibitors should be continued
    - beta-blockers should only be stopped if the patient has heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock

+ Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

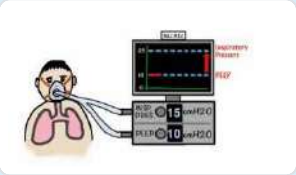
Links

NICE 👍 6 👎 9

2014 Acute heart failure: diagnosis and management

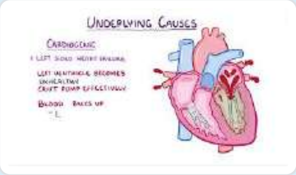
Suggest link Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 👍 17 👎 0


UNDERLYING CAUSES
CARDIOGENIC

Pulmonary oedema

Osmosis - YouTube 👍 4 👎 0

Report broken media

Score: **19.4%**

1 ✗
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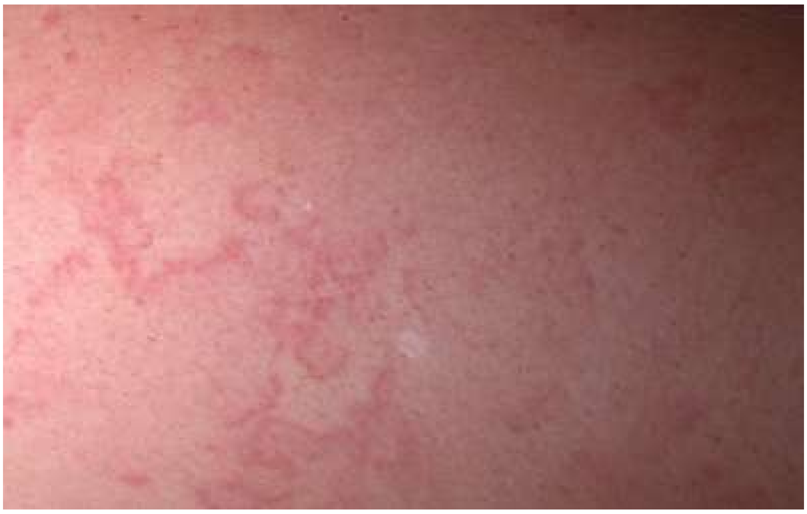
A 10-year-old girl is reviewed. Her mother describes her as being 'generally unwell'. For the past week she has been having joint pains and fever. Her mother also describes episodes of her making jerky, irregular movements. She recently went on holiday to the Lake District and had a severe sore throat around 4 weeks ago.

On examination her temperature is 37.9ºC, pulse 90/min, blood pressure 100/62 mmHg. Auscultation of the heart is unremarkable. A rash is noted on her torso:



© Image used on license from DermNet NZ

What does the rash most likely represent?

○ Erythema marginatum

○ Erythema multiforme

○ Erythema chronicum migrans

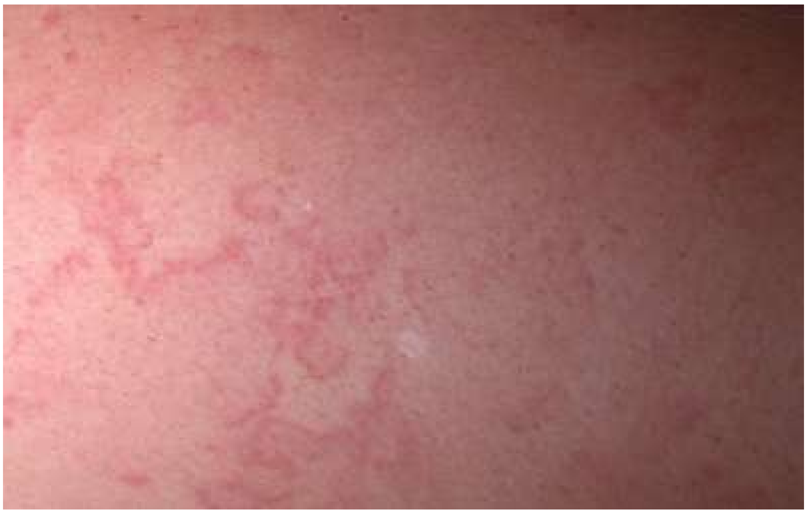
○ Janeway lesions

○ Erythema toxicum

Submit answer

A 10-year-old girl is reviewed. Her mother describes her as being 'generally unwell'. For the past week she has been having joint pains and fever. Her mother also describes episodes of her making jerky, irregular movements. She recently went on holiday to the Lake District and had a severe sore throat around 4 weeks ago.

On examination her temperature is 37.9ºC, pulse 90/min, blood pressure 100/62 mmHg. Auscultation of the heart is unremarkable. A rash is noted on her torso:



© Image used on license from DermNet NZ

What does the rash most likely represent?

Erythema marginatum	73%
Erythema multiforme	12%
Erythema chronicum migrans	11%
Janeway lesions	2%
Erythema toxicum	2%

The underlying diagnosis is rheumatic fever. This is supported by the recent sore throat, chorea (jerk, irregular movements) and polyarthralgia.

Erythema toxicum is a non-specific rash seen in newborns.

👍 👎 Discuss (5) Improve

Next question >

Rheumatic fever ★

Rheumatic fever develops following an immunological reaction to a recent (2-4 weeks ago) *Streptococcus pyogenes* infection.

Pathogenesis

- *Streptococcus pyogenes* infection → activation of the innate immune system leading to antigen presentation to T cells
- B and T cells produce IgG and IgM antibodies and CD4+ T cells are activated
- there is then a cross-reactive immune response (a form of type II hypersensitivity) thought to be mediated by molecular mimicry
- the cell wall of *Streptococcus pyogenes* includes M protein, a virulence factor that is highly antigenic. It is thought that the antibodies against M protein cross-react with myosin and the smooth muscle of arteries
- this response leads to the clinical features of rheumatic fever
- Aschoff bodies describes the granulomatous nodules found in rheumatic heart fever

Diagnostic criteria

Diagnosis is based on evidence of recent streptococcal infection accompanied by:

- 2 major criteria
- 1 major with 2 minor criteria

Evidence of recent streptococcal infection

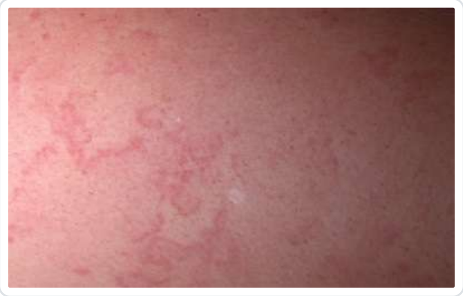
- raised or rising streptococci antibodies,
- positive throat swab
- positive rapid group A streptococcal antigen test

Major criteria

- erythema marginatum
- Sydenham's chorea: this is often a late feature
- polyarthritis
- carditis and valvulitis (eg, pancarditis)
    - The latest iteration of the Jones criteria (published in 2015) state that rheumatic carditis cannot be based on pericarditis or myocarditis alone and that there must be evidence of endocarditis (the clinical correlate of which is valvulitis which manifests as a regurgitant murmur)
- subcutaneous nodules

Minor criteria

- raised ESR or CRP
- pyrexia
- arthralgia (not if arthritis a major criteria)
- prolonged PR interval



Management

Outline of management

- antibiotics: oral penicillin V
- anti-inflammatories: NSAIDs are first-line
- treatment of any complications that develop e.g. heart failure

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ 👍 8 👎 5

Rheumatic fever

Suggest link

Report broken link

Media

RHEUMATIC FEVER
&
HEART DISEASE
Osmosis.org

Rheumatic fever & heart disease

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **19%**

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A 22-year-old man is brought to the emergency department after being granted asylum in the UK. Immigration officials had been concerned about the patient's well-being immediately on his arrival in the country. The patient reported feeling progressively more unwell over the previous 3 months after he had fled from his village in Syria. The primary symptoms reported by the patient were fevers, severe night sweats and profound fatigue. In addition, the patient had found that his exercise tolerance has greatly reduced in the previous four weeks and he became markedly breathless on minimal exertion.

The patient explained that he had experienced a prolonged period of inadequate food and shelter due to the war in his home country and while travelling across Europe. The patient had previously lived in a small village in Syria and had worked as a sheep farmer. Prior to fleeing his home the patient had regularly consumed food products made with unpasteurised sheep's milk. The patient had previously been in good physical health and had suffered no significant medical problems during his life.

On general examination, the patient appeared unwell and diaphoretic. The patient's sweat was noted to have an unusually unpleasant odour. Examination of the cardiovascular system identified multiple splinter haemorrhages under the patient's fingernails and toenails. Jugular venous pressure was elevated by 3-4 cm and pitting oedema was present to the patient's knees. A harsh systolic murmur was heard over the aortic area on auscultation. Bilateral basal crackles were heard on auscultation of the patient's lungs. The patient's spleen was palpable on examination of the abdominal system. No palpable lymphadenopathy was identified.

Please see the below table for results of initial investigations.

Haemoglobin	123 g / dL
Mean cell volume	80.3 fl
Lymphocytes	4.9 x 10>3 / microlitre (reference 1.5-4.0)
Platelets	109 x 10>3 / microlitre
Urea	8.9 mmol / L

Creatinine	126 micromols / L
Sodium	137 mmol / L
Potassium	4.1 mmol / L
Erythrocyte sedimentation rate	102 mm / h (reference 0-35)
C-reactive protein	99 mg / L (reference < 7)
Urinalysis	Positive for blood +++
Chest x-ray	Upper lobe blood diversion; generalised patchy airspace shadowing; bilateral shallow pleural effusions
ECG	Sinus rhythm; borderline right bundle branch block
Focused bedside transthoracic echocardiogram	0.6 cm vegetation on aortic valve; evidence of moderate-severe aortic stenosis; ejection fraction 30 %

Which of the following organisms is the most likely cause of the patient's presentation?

- ○ *Clostridium burnetti* ×
- ○ *Bartonella quintana* ×
- ○ *Haemophilus parainfluenzae* ×
- ○ *Kingella kingae* ×
- ○ *Brucella melitensis* ×

Submit answer

Reference ranges

Score: **19%**

1 ×

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ECG	Sinus rhythm; borderline right bundle branch block
Focused bedside transthoracic echocardiogram	0.6 cm vegetation on aortic valve; evidence of moderate-severe aortic stenosis; ejection fraction 30 %

Which of the following organisms is the most likely cause of the patient's presentation?

Clostridium burnetti	9%
Bartonella quintana	12%
Haemophilus parainfluenzae	2%
Kingella kingae	5%
Brucella melitensis	72%

The patient presents with clear symptoms, signs and investigation results consistent with aortic valve endocarditis and secondary congestive cardiac failure. It is likely that the patient's prolonged exposure to inadequate living conditions has weakened his immune system allowing the development of an opportunistic infection.

Of the available options, *Brucella melitensis* is the most likely to be the causative microorganism. Endocarditis - most commonly of the aortic valve - is a rare complication of Brucella; however, it is responsible for the majority of mortality from this disease. Of the different *Brucella* species known to cause endocarditis *Brucella melitensis* is known to be associated with the most severe complications.

Brucellosis is the most likely diagnosis in this patient due to his history of consumption of unpasteurised milk products and close contact with livestock from an endemic region. The clinical sign of malodorous sweat is considered to be nearly pathognomonic for Brucellosis. In addition, the patient's blood results are typical for the condition featuring lymphocytosis and thrombocytopenia.

The patient requires blood cultures and serological testing to confirm the diagnosis. Given the severity of the patient's condition, he would require treatment with empirical antibiotics with consideration of aortic valve replacement at an appropriate point in the future.

The other available answers are all organisms known to be rare causes of endocarditis that usually will not be identified without specialist microbiological investigations. There is no information in the case presentation that makes any of these microorganisms a more likely causative agent of endocarditis than *Brucella melitensis*.

Ramin B, MacPherson P. Human brucellosis. *BMJ* 2010;341:c4545.

👍 👎 Discuss (7) Improve

Next question >

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
  - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
  - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis
    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*
    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link

Report broken link

Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
DUKE'S CRITERIA
JANEWAY LESIONS
OSLER NODES

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

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Score: **18.8%**

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Question 65 of 328

A 64-year-old hypertensive male presents to the emergency department with a history of sudden onset tearing chest pain. He is a chronic smoker with a 25 pack year history. He takes valsartan 160mg daily and amlodipine 5mg daily.

On examination, he is distressed and in severe pain. Blood pressure is 200/120 mmHg. The peripheral pulses are weak and his heart rate is 125 bpm.

Sublingual nitrates and oral aspirin are given to him in the emergency department and his twelve-lead ECG is performed which reveals tachycardia, left ventricular strain and deep S waves in lead V1-V3 and tall R waves in V4-V6.

Chest x-ray shows widening of the mediastinum with an irregular aortic contour.

Contrast-enhanced CT scan of the chest reveals an aortic intimal flap distal to left subclavian artery.

Which of the following is the most appropriate treatment option for this patient?

- ○ Blood pressure control with IV beta-blockers ×
- ○ Percutaneous stent insertion ×
- ○ Surgical repair ×
- ○ Blood pressure control with IV hydralazine ×
- ○ Continuous infusion of isosorbide dinitrate ×

Submit answer

Reference ranges

Score: **18.8%**

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← Question 65 of 328 ✗ →

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Chest x-ray shows widening of the mediastinum with an irregular aortic contour.

Contrast-enhanced CT scan of the chest reveals an aortic intimal flap distal to left subclavian artery.

Which of the following is the most appropriate treatment option for this patient?

Blood pressure control with IV beta-blockers	76%
Percutaneous stent insertion	1%
Surgical repair	15%
Blood pressure control with IV hydralazine	7%
Continuous infusion of isosorbide dinitrate	1%

The diagnosis in this question is aortic dissection type B (distal to the left subclavian artery), the management of which is blood pressure control. The most appropriate drugs used are IV beta-blockers.

Aortic dissections can be classified according to the anatomic site of the dissection. Type A involves the aortic arch and the aortic valve while type B involves the descending thoracic aorta distal to the left subclavian artery. (Stanford Classification)

Stanford types A and B are further sub-classified according to the De Bakey classification.

Stanford Type A constitutes De Bakey I (extending to the abdominal aorta) and De Bakey II (localised to the ascending aorta). Stanford type B is synonymous with De Bakey type III.

The importance of these classifications is the difference in treatment. Universally, blood pressure control in mandatory with a target reduction in systolic BP to 120mmHg. Type A dissection should undergo surgery (aortic arch replacement) if the patient is fit enough since medical management alone carries a high risk of mortality (50% within 2 weeks). Type B dissections carry a better prognosis with 89% survival at one month and should thus initially be managed medically, with surgical interventions only if complications develop.

Indications for endovascular stenting include:
- Rapidly expanding dissections (>1cm per year)
- Critical diameter (>5.5cm)
- Refractory pain
- Malperfusion syndrome
- Blunt chest trauma
- Penetrating aortic ulcers

Discuss (9) | Improve

Next question

Aortic dissection: investigation and management

Classification

Stanford classification

- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally

Investigation

It's important to remember that patients may present acutely and be clinically unstable. The choice of investigations will have to take account of this.

Chest x-ray

- widened mediastinum

CT angiography of the chest, abdomen and pelvis is the investigation of choice

- suitable for stable patients and for planning surgery
- a false lumen is a key finding in diagnosing aortic dissection

Transoesophageal echocardiography (TOE)

- more suitable for unstable patients who are too risky to take to CT scanner

Management

Type A

- surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention

Type B*

- conservative management
- bed rest
- reduce blood pressure IV labetalol to prevent progression

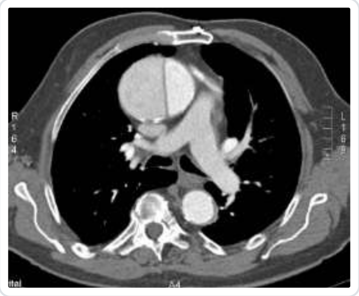
Complications

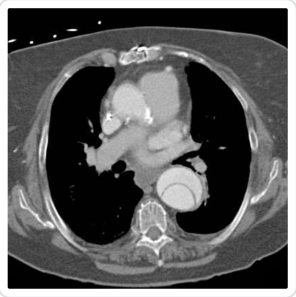
Complications of backward tear

- aortic incompetence/regurgitation
- MI: inferior pattern is often seen due to right coronary involvement

Complications of a forward tear

- unequal arm pulses and BP
- stroke
- renal failure





*endovascular repair of type B aortic dissection may have a role in the future

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

American Heart Association 👍 13 👎 13

2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease

Suggest link

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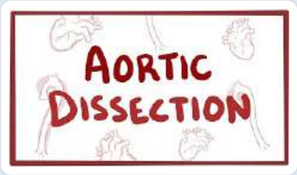
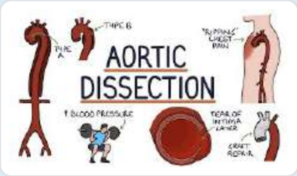
Media


AORTIC
DISSECTION

Aortic dissection

Osmosis - YouTube 👍 9 👎 0


TYPE B
TYPE A
AORTIC
DISSECTION
"RIPPING" CHEST PAIN
↑ BLOOD PRESSURE
TEAR OF INTIMA LAYER
GRAFT REPAIR

Understanding Aortic Dissection

Zero To Finals - YouTube 👍 1 👎 0

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Score: **18.5%**

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Question 66 of 328

A 32-year-old woman with pyrexia of unknown origin is admitted to the medical assessment unit.

Examination reveals a pale patient with a new cardiac murmur, evidence of splinter haemorrhages and splenomegaly. She looks unwell.

She has no past medical history, takes no regular medication and has no allergies.

The patient has high inflammatory markers and positive blood cultures which are growing Gram-positive cocci in clusters.

What is the single most appropriate antibiotic treatment?

- ○ IV amoxicillin ×
- ○ IV flucloxacillin ×
- ○ IV gentamicin ×
- ○ IV vancomycin ×
- ○ Oral doxycycline ×

Submit answer

Reference ranges

Score: **18.5%**

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Question 66 of 328 ✓

A 32-year-old woman with pyrexia of unknown origin is admitted to the medical assessment unit.

Examination reveals a pale patient with a new cardiac murmur, evidence of splinter haemorrhages and splenomegaly. She looks unwell.

She has no past medical history, takes no regular medication and has no allergies.

The patient has high inflammatory markers and positive blood cultures which are growing Gram-positive cocci in clusters.

What is the single most appropriate antibiotic treatment?

IV amoxicillin	17%
IV flucloxacillin	75%
IV gentamicin	1%
IV vancomycin	6%
Oral doxycycline	0%

IV flucloxacillin is the treatment of choice for native valve endocarditis caused by staphylococci

Important for me Less important

The most likely diagnosis here is infective endocarditis given the widespread clinical signs and the positive blood culture is most likely a *Staphylococcus aureus*, which is often described as Gram-positive cocci in clusters before full identification is completed.

The most appropriate antibiotic, therefore, is **IV flucloxacillin** which will adequately treat *S aureus*. This is the recommended treatment for any native valve staphylococcal endocarditis and is the correct answer in this case.

IV amoxicillin is incorrect. Although it is the recommended empirical antibiotic for endocarditis, it is incorrect to use if there is a known staphylococcal organism in the blood culture.

IV gentamicin is often added to amoxicillin in native valve endocarditis but it is unnecessary as the organism has been identified in a blood culture.

You may see a patient with a prosthetic valve where you would need to consider using **IV vancomycin** to cover an MRSA, however, in this case, the patient has no medical history so this is not correct.

Oral doxycycline would treat *S aureus* but is inappropriate as IV antibiotics are always required in infective endocarditis.

👍 | 👎 | Discuss | Improve

Next question >

Infective endocarditis: prognosis and management ★

Poor prognostic factors
- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism
- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve - amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis - vancomycin + low-dose gentamicin If prosthetic valve - vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA - vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA - vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic - vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic - vancomycin + low-dose gentamicin

Indications for surgery

- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

+ Q 123

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Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube

👍 4 👎 0

[Endocarditis](#)

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **19.7%**

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Question 67 of 328

A 55-year-old obese man with a history of hypertension, diabetes and angina develops central crushing chest pain whilst gardening alone. He is short of breath and clammy. He takes two puffs of his glyceryl trinitrate spray but has only mild relief from this.

He calls an ambulance, but before the ambulance arrives he suffers a cardiac arrest. The ambulance crew commence cardiopulmonary resuscitation (CPR). Heart monitoring shows him to be in ventricular tachycardia and after a DC shock and a further round of CPR, he recovers cardiac output.

He is taken to the local hospital intensive care unit where he remains unconscious. The doctors explain to his family that because of the time between his cardiac arrest and CPR commencing, his brain function may not recover well.

What is the most reliable indicator of this patient's prognosis at 72 hours post-arrest?

- ○ Electroencephalogram
- ○ Glasgow coma scale motor score
- ○ Pupillary reflex
- ○ Somatosensory evoked potential
- ○ Gag reflex

Submit answer

Reference ranges

A 55-year-old obese man with a history of hypertension, diabetes and angina develops central crushing chest pain whilst gardening alone. He is short of breath and clammy. He takes two puffs of his glyceryl trinitrate spray but has only mild relief from this.

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He is taken to the local hospital intensive care unit where he remains unconscious. The doctors explain to his family that because of the time between his cardiac arrest and CPR commencing, his brain function may not recover well.

What is the most reliable indicator of this patient's prognosis at 72 hours post-arrest?

🟥 Electroencephalogram	19%
Glasgow coma scale motor score	21%
🟩 Pupillary reflex	34%
Somatosensory evoked potential	17%
Gag reflex	8%

Ability to predict poor outcome (death or severe disability) in a comatosed patient followings cardiac arrest is important for decision making regarding withdrawal of treatments as well as communicating with families.

No clinical neurological signs have been shown to correlate reliably with poor outcomes when measured less than 24 hours after cardiac arrest.

Work by Rittenberger and colleagues (2010) looked at patients who suffered ventricular tachycardia or ventricular fibrillation cardiac arrests. They found that GCS motor score did not correlate with cardiac arrest outcome when measured at 24 or 72 hours. However, lack of pupillary light or corneal reflex at 72 hours was a reliable predictor of death.

Vestibulo-ocular reflexes are also used but are less reliable. No biochemical, imaging or neurophysiological studies are currently recommended for the assessment of prognosis.

References:

Resuscitation Council (UK). Advanced Life Support. 6th Edition. London: Resuscitation Council (UK); 2011.

Rittenberger JC, Sangl J, Wheeler M, Guyette FX, Callaway CW. Association between clinical examination and outcome after cardiac arrest. Resuscitation 2010; 81: 11281132.

👍 👎 Discuss (10) Improve

Next question ›

Adult advanced life support ★

The following is based on the 2021 Resus Council guidelines. Please see the link for more details, below is only a very brief summary of key points.

It should be remembered that the algorithm divides patients into those with:
- 'shockable' rhythms: ventricular fibrillation/pulseless ventricular tachycardia (VF/pulseless VT)
- 'non-shockable' rhythms: asystole/pulseless-electrical activity (asystole/PEA)

Major points include:
- chest compressions
    - the ratio of chest compressions to ventilation is 30:2

    - chest compressions are now continued while a defibrillator is charged
- defibrillation
    - a single shock for VF/pulseless VT followed by 2 minutes of CPR
    - if the cardiac arrested is witnessed in a monitored patient (e.g. in a coronary care unit) then the 2015 guidelines recommend 'up to three quick successive (stacked) shocks', rather than 1 shock followed by CPR
- drug delivery
    - IV access should be attempted and is first-line
    - if IV access cannot be achieved then drugs should be given via the intraosseous route (IO)
    - delivery of drugs via a tracheal tube is no longer recommended
- adrenaline
    - adrenaline 1 mg as soon as possible for non-shockable rhythms
    - during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock
    - repeat adrenaline 1mg every 3-5 minutes whilst ALS continues
- amiodarone
    - amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.
    - a further dose of amiodarone 150 mg should be given to patients who are in VF/pulseless VT after 5 shocks have been administered
    - lidocaine used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead
- thrombolytic drugs
    - should be considered if a pulmonary embolus is suspected
    - if given, CPR should be continued for an extended period of 60-90 minutes

Resuscitation Council UK
GUIDELINES 2021
Adult advanced life support

Other points

- atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA)

- following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

Reversible causes of cardiac arrest:

The 'Hs'	The 'Ts'
• Hypoxia • Hypovolaemia • Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders • Hypothermia	• Thrombosis (coronary or pulmonary) • Tension pneumothorax • Tamponade - cardiac • Toxins

+ Q 123

Next question >

B I A T↕

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Links

Resuscitation Council 👍 32 👎 14

2021 Basic Life Support guidelines

Resuscitation Council 👍 58 👎 25

2021 Advanced Life Support guidelines

Suggest link Report broken link

Media

RC (UK) Cardiac Arrest Management Demo

Resuscitation Council (UK) - YouTube 👍 16 👎 2

Report broken media

Score: **19.4%**

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Question 68 of 328

A 74-year-old gentleman presents with oedema. He also states that his urine has been frothy over the last few weeks. He has no past medical history and is on no regular medications.

On examination he has pitting oedema up to the mid thighs. His JVP is 8cm from the sternal angle, and rises with inspiration. His pulse is irregularly irregular, and ECG shows fast atrial fibrillation with a rate of 145 bpm. He is otherwise systemically well with a BP of 135/85mm Hg. On neurological exam you note weakness and a sensory deficit in the distribution of the right ulnar nerve and left posterior tibial nerve.

You administer a loading dose of 1mg IV digoxin over 2 hours. Shortly after this the patient deteriorates and loses consciousness. A repeat ECG shows ventricular tachycardia, which then develops into ventricular fibrillation. Unfortunately the patient did not survive resuscitation attempts.

What is the most likely underlying disease?

- ○ Hypertrophic obstructive cardiomyopathy ×
- ○ AL amyloidosis ×
- ○ Brugada disease ×
- ○ AA amyloidosis ×
- ○ Cryoglobulinaemia ×

Submit answer

Reference ranges

Score: **19.4%**

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← Question 68 of 328 ✗ →

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You administer a loading dose of 1mg IV digoxin over 2 hours. Shortly after this the patient deteriorates and loses consciousness. A repeat ECG shows ventricular tachycardia, which then develops into ventricular fibrillation. Unfortunately the patient did not survive resuscitation attempts.

What is the most likely underlying disease?

Hypertrophic obstructive cardiomyopathy	9%
AL amyloidosis	47%
Brugada disease	7%
AA amyloidosis	30%
Cryoglobulinaemia	6%

Digoxin administration is not recommended in cardiac amyloidosis owing to a higher risk of digoxin toxicity, as the drug binds avidly to amyloid fibrils

Important for me Less important

The presence of Kussmauls sign (JVP which rises on inspiration) and significantly raised JVP is suggestive of constrictive pericarditis or restrictive cardiomyopathy. Together with the presence of nephrotic syndrome, and mononeuritis multiplex, one must consider amyloidosis.

AA amyloidosis occurs due to chronic inflammation e.g. rheumatoid arthritis. There is no evidence of a past medical history that would predispose to AA amyloidosis. Furthermore, cardiac manifestations are very rare in AA amyloidosis. This is in contrast to AL amyloidosis, which occurs due to light chain disease, where cardiac manifestations are very common.

Digoxin administration is not recommended in cardiac amyloidosis owing to a higher risk of digoxin toxicity, as the drug binds avidly to amyloid fibrils. In this case, administration of digoxin has led to cardiac arrhythmia due to the enhanced effects of digoxin on an amyloid heart.

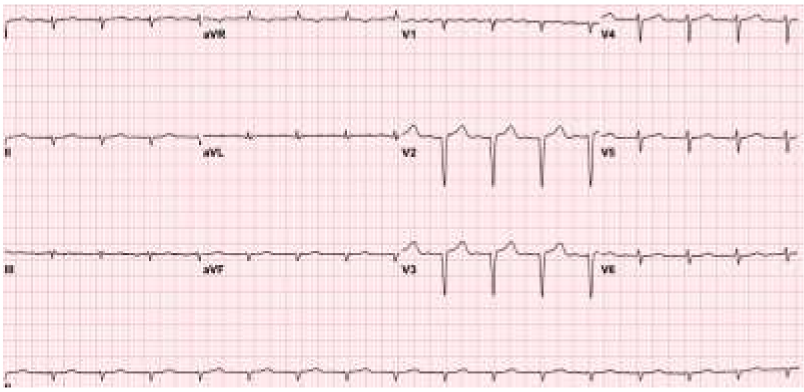
👍 👎 💬 Discuss (5) Improve

Next question >

Amyloidosis: cardiac ★

Cardiac amyloidosis, also known as amyloid cardiomyopathy, is a rare disorder characterized by the extracellular deposition of insoluble amyloid fibrils in the myocardium, leading to progressive heart failure and conduction abnormalities. Cardiac amyloidosis can occur in isolation or as part of systemic amyloidosis, which affects multiple organs.

Investigations
- ECG: typically shows low-voltage complexes with poor R wave progression in the chest leads (a pseudoinfarction pattern).
- echocardiogram: increased myocardial echogenicity (granular sparkling or 'global speckled' appearance)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123

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Textbooks

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Links

Journal of the American Heart Association 6 0

Updates in Cardiac Amyloidosis: A Review

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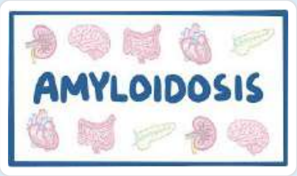
Media


AMYLOIDOSIS

Amyloidosis

Osmosis - YouTube 👍 17 👎 2

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Score: **19.1%**

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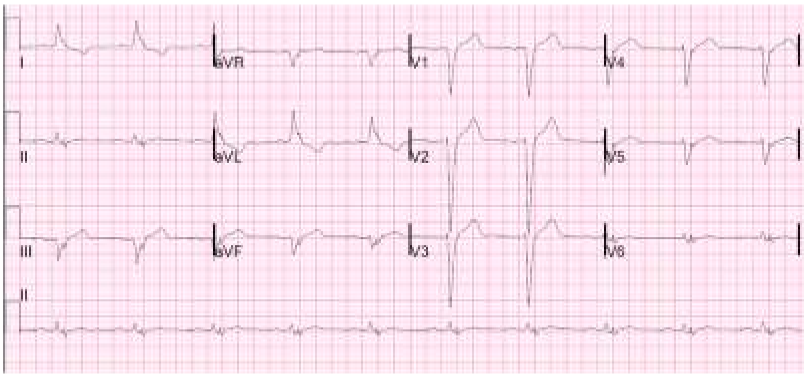
A 49-year-old man presents to the emergency department of a tertiary centre with a 4-hour history of central chest pain. There is no radiation of pain, but he reports to be feeling breathless and light-headed. Other than a recent viral upper respiratory tract infection, he has been well in himself recently. His past medical history includes hypertension, hyperlipidaemia and a post-operative deep vein thrombosis 2 years ago. Current medications include ramipril, amlodipine and atorvastatin and he denies any allergies.

On examination, he appears clammy to touch. His chest is clear on auscultation, although he is mildly tachypnoeic with saturations of 94%. Heart sounds are normal and his peripheral pulses are intact. He has a soft and non-tender abdomen.

An ECG is recorded, as shown below:



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Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

- ○ Discharge with NSAIDs
- ○ Fibrinolysis
- ○ Insulin and dextrose infusion
- ○ Percutaneous coronary intervention

○ Rivaroxaban ×

Submit answer

Reference ranges

Score: **19.1%**

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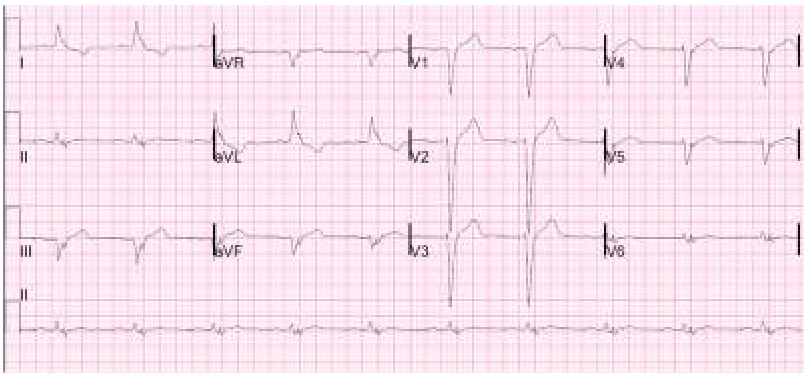
On examination, he appears clammy to touch. His chest is clear on auscultation, although he is mildly tachypnoeic with saturations of 94%. Heart sounds are normal and his peripheral pulses are intact. He has a soft and non-tender abdomen.

An ECG is recorded, as shown below:



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Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

Discharge with NSAIDs	25%
Fibrinolysis	6%
Insulin and dextrose infusion	3%
Percutaneous coronary intervention	60%

Rivaroxaban 6%

This patient's ECG shows a left bundle branch block (LBBB) with broadened QRS complexes (> 120ms), a dominant S wave in V1 (creating 'W' impression) and broad monophasic R waves in leads I, aVL, V5 and V6 (creating 'M' impression). In patients with an ECG showing LBBB, it is important to establish whether or not this is a new finding as a new presentation of LBBB is concerning for myocardial ischaemia. In combination with a history of chest pain, shortness of breath, and being clammy to touch, it is likely that this patient is having a myocardial infarction. For patients presenting to the hospital within 12 hours of a STEMI with the availability of primary percutaneous coronary intervention (PCI) within 2 hours, PCI is the most appropriate and definitive management of this patient where the obstructed artery can be stented. As this patient has presented within 4 hours to a tertiary centre, it is likely that PCI can be achieved and is, therefore, the correct answer.

It would be inappropriate to discharge this patient with analgesia as a new finding of LBBB and a clinical history of chest pain is highly suggestive of myocardial ischaemia and needs urgent treatment.

Fibrinolysis is an alternative option for managing acute coronary syndrome. However, if PCI is available, it should be prioritised as the most appropriate treatment.

Insulin and dextrose are used in the management of hyperkalaemia. ECG changes in hyperkalaemia commonly include peaked T waves. However, in severe hyperkalaemia, ECG changes can include bundle branch blocks including LBBB. This is much rarer than myocardial ischaemia and given the history of chest pain, shortness of breath and sweating, an acute coronary syndrome is the more likely cause for which PCI is indicated.

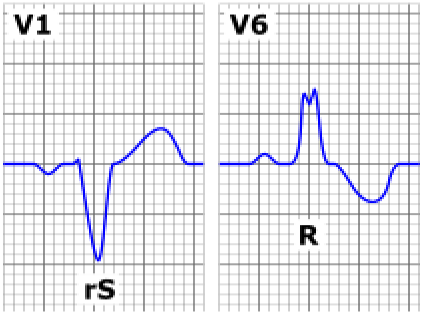
Rivaroxaban is used in the treatment of a pulmonary embolism (PE). Although this patient has a higher risk of venous thromboembolism with a previous DVT, it is very unlikely for a PE to cause LBBB.

👍 👎 Discuss (10) Improve

Next question ›

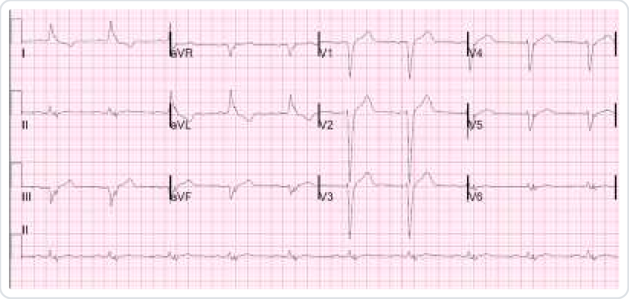
ECG: left bundle branch block

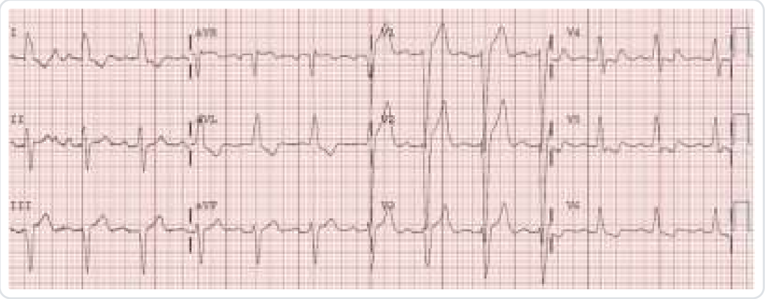
The diagram below shows the typical features of left bundle branch block (LBBB):



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6





New LBBB is always pathological. Causes of LBBB include:

- myocardial infarction
  - diagnosing a myocardial infarction for patients with *existing* LBBB is difficult
  - rhe Sgarbossa criteria can help with this - please see the link for more details
- hypertension
- aortic stenosis
- cardiomyopathy

- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

123

Next question

Textbooks

High-yield textbook

Extended textbook

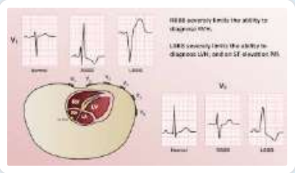
Links

Life in the Fast Lane 👍 33 👎 16

Sgarbossa criteria

Suggest link Report broken link

Media



Intro to EKG Interpretation - Bundle Branch Blocks

Strong Medicine - YouTube 👍 6 👎 2

Bundle branch block

Osmosis - YouTube 👍 0 👎 3

Report broken media

Score: **18.8%**

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Question 70 of 328

An 84-year-old gentleman is admitted to hospital with a short history of episodes of loss of consciousness. The patient has no clear recollection of the events, but his family informs you that they have not identified any specific pattern to them. Most of them have been unwitnessed, with the exception of the last one, where according to his wife he became pale, his eyes rolled backwards and fell on the chair. Some minor flickering was noted. Once he regained consciousness he was briefly confused and anxious, but soon realised where he was. Cardiovascular examination was unremarkable, with no postural hypotension but a blood pressure of 103/55 mmHg, and no focal neurology was isolated. His bloods were unremarkable.

His ECG was that of sinus rhythm: 67/min with Right Bundle Branch Block (RBBB). You see from a previous echocardiogram in his notes, that he has a degree of heart failure with an ejection fraction of 45%. A 72 hour holter monitor revealed no significant pauses.

Which action would you take next?

- ○ Arrange MRI head
- ○ Arrange EEG
- ○ CRT-D placement
- ○ Arrange insertion of Reveal Device
- ○ Arrange performance of Carotid sinus and Tilt-test

Submit answer

Reference ranges

Score: **18.8%**

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Question 70 of 328 ✗

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Which action would you take next?

Arrange MRI head	8%
Arrange EEG	11%
CRT-D placement	19%
Arrange insertion of Reveal Device	24%
Arrange performance of Carotid sinus and Tilt-test	39%

In patient with BBB and unexplained syncope but an ejection fraction >35% proceed with further testing (e.g. carotid sinus massage, electrophysiological studies) prior to initiation of management

Important for me Less important

As per the ESC Guidelines on Cardiac Pacing and Cardiac Resynchronization therapy (2013) it is important to exclude any other probable causes of collapse/syncope prior to installing any device. It is important to note that 'Less than half of the patients with BBB and syncope have a final diagnosis of cardiac syncope, albeit the probability is different among different type of BBB'. Given his symptomatology and the examination findings alongside the 72-hour holter monitor this is unlikely to be purely cardiogenic therefore further investigation is needed. With his age and presentation, autonomic dysfunction could be the cause, therefore carotid sinus and tilt-test may provide the answer. An MRI head and an EEG remain plausible options, but given the lack of solid seizure-like activity, can be arranged later on, provided that no answer is given by other tests.

👍 👎 Discuss (8) Improve

Next question >

Pacemaker: permanent ★

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:

- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):
- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):
- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological

indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):
- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:
- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:
- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):
- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial

and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

+ Q 123

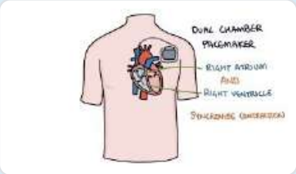
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


DUAL CHAMBER PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **18.6%**

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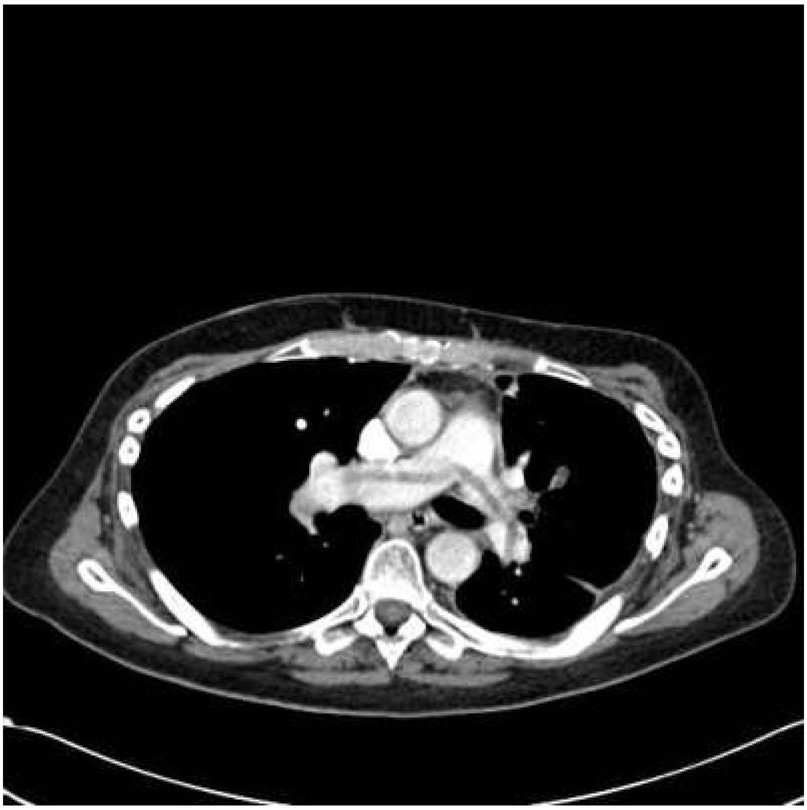
Question 71 of 328

A 72-year-old man is admitted to hospital with shortness-of-breath. On examination his pulse is 96/min, BP 100/64 mmHg, respiratory rate 20/min, temperature 37.5ºC, oxygen saturations 96% on room air. A 12-lead ECG shows sinus rhythm, at a rate of 94/min with no diagnostic ST-T changes. The troponin I level is < 0.05 µg/L. A CT chest (with contrast) is ordered:



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What is the most likely diagnosis?

- ○ Superior vena cava obstruction
- ○ Aortic dissection (Stanford type A)
- ○ Infective endocarditis
- ○ Aortic dissection (Stanford type B)
- ○ Pulmonary embolism

Submit answer

Reference ranges

Score: **18.6%**

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Question 71 of 328 ✗

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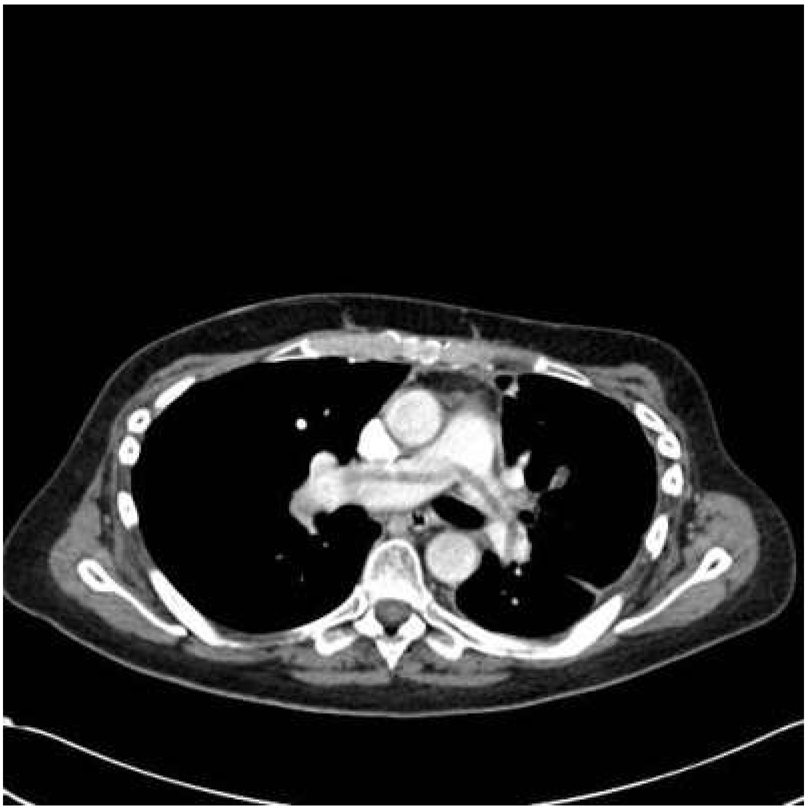

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What is the most likely diagnosis?

Superior vena cava obstruction	2%
Aortic dissection (Stanford type A)	22%
Infective endocarditis	1%
Aortic dissection (Stanford type B)	11%
Pulmonary embolism	64%

The CT shows a large saddle embolus where the pulmonary trunk splits to form the right and left pulmonary arteries.

Most image-based questions can be answered without looking at the image. If we therefore concentrate on the history a few clues point towards a pulmonary embolism (PE). The first is tachypnoea. Around 95% of patients with a PE have a respiratory rate > 16/min. Tachypnoea is not as common in any of the other diagnoses. A low-grade temperature is also an under appreciated sign of pulmonary embolism. This could of course be consistent with infective endocarditis but there are no other features to support this diagnosis.

👍 👎 Discuss (5) Improve

Next question >

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:

- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

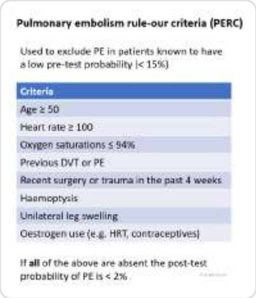
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
    - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3

Clinical feature	Points
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores

- PE likely - more than 4 points
- PE unlikely - 4 points or less

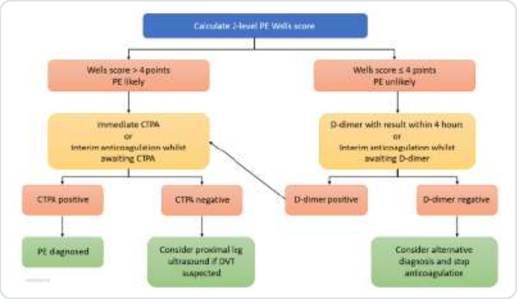
If a PE is 'likely' (more than 4 points)

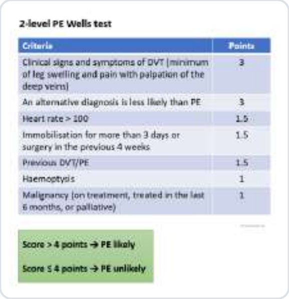
- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
  - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
  - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
  - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
  - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed

  - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points PE likely
Wells score ≤ 4 points PE unlikely
Immediate CTPA or Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours or Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

Some other points

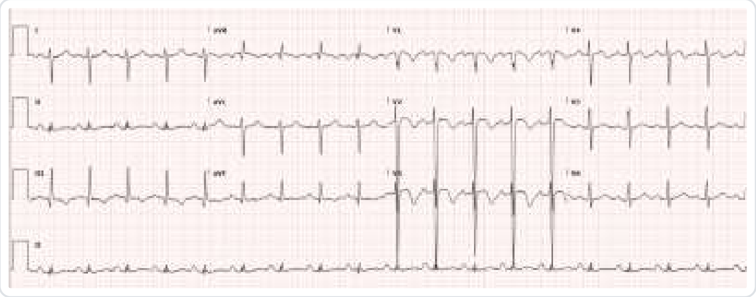
D-dimers

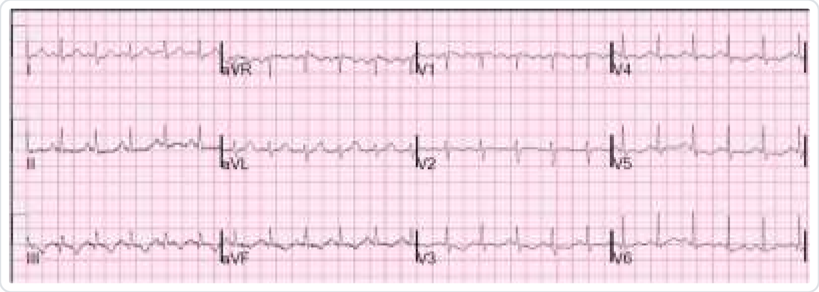
- sensitivity = 95-98%, but poor specificity

- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen

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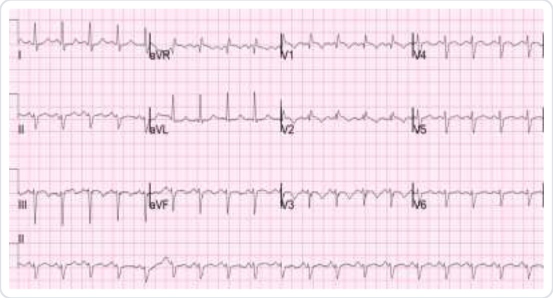
I
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Chest x-ray

- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

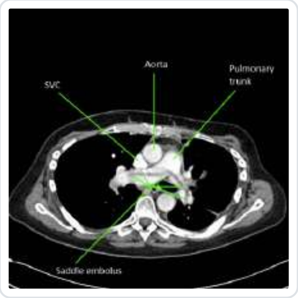
V/Q scan

- sensitivity of around 75% and specificity of 97%

- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
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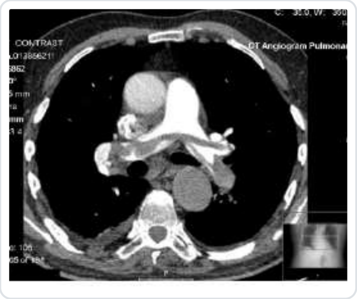
Saddle embolus


CONTRAST
CT Angiogram Pulmonar

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 13 👎 10

The ECG in Pulmonary Embolism

MDcalc 👍 15 👎 8

Wells' Criteria for Pulmonary Embolism

MDcalc 👍 17 👎 8

PERC Rule for Pulmonary Embolism

NICE 👍 20 👎 9

2020 Venous thromboembolism guidelines

Suggest link Report broken link

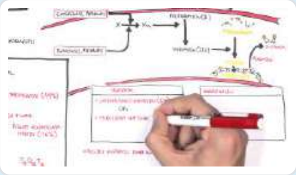
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

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Question 72 of 328

A 19-year-old woman is being reviewed in the cardiology clinic for recurrent syncopal attacks in the last 2 years. The attacks mainly occur during or after strenuous exercise.

Her vital signs are a temperature of 37 °C, blood pressure 105/65 mmHg, pulse 83 beats/minute, and oxygen saturation 98% on room air. Cardiac examination is unremarkable. She has thickened skin over her palms and soles. An electrocardiogram (ECG) shows deep t-wave inversions in V1 to V3.

What is the most appropriate anti-arrhythmic drug for this patient?

○ Bisoprolol ×

○ Digoxin ×

○ Magnesium sulphate ×

○ Metoprolol ×

○ Sotalol ×

Submit answer

Reference ranges

Score: **18.3%**

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What is the most appropriate anti-arrhythmic drug for this patient?

Bisoprolol	23%
Digoxin	4%
Magnesium sulphate	8%
Metoprolol	7%
Sotalol	58%

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Important for me Less important

Syncopal attacks precipitated by exercise and deep T-wave inversions from V1 to V3 point towards arrhythmogenic right ventricular cardiomyopathy (ARVC). ARVC, as described in this scenario, can occur with hereditary Naxos disease in which woolly hair and palmoplantar skin thickening can be seen. **Sotalol** is the most effective anti-arrhythmic drug for patients with ARVC. Sotalol is superior to amiodarone in preventing and treating ventricular arrhythmias in ARVC.

Bisoprolol is incorrect because the anti-arrhythmic properties of bisoprolol are not equivalent to sotalol in preventing arrhythmias in

ARVC. Bisoprolol is a widely used rate-controlling agent and less frequently used as an anti-arrhythmic.

Digoxin is unlikely to benefit this patient with ARVC. Digoxin is used as an anti-arrhythmic in patients with concomitant heart failure.

Magnesium sulphate has no role in treating ARVC. It is an anti-arrhythmic of choice in torsades de pointes.

The patient in this scenario has features similar to hypertrophic cardiomyopathy (HCM), in which **metoprolol** is the first-line drug of choice. The skin keratosis described in this scenario makes Naxos disease and ARVC more likely.

👍 👎 Discuss Improve

Next question ›

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology
- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation
- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 13 7

Arrhythmogenic right ventricular cardiomyopathy ECG

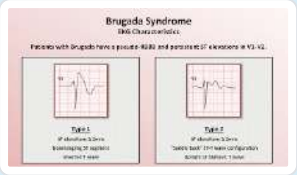
Suggest link Report broken link

Media

CARDIOLOGY
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome
EKG Characteristics
Type 1
Type 2

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

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Score: **18.1%**

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Question 73 of 328

A 72-year-old male presents with a five hour history of left sided chest pain, radiating to his left arm. He also complains of sweatiness in both his hands and has vomited three times in the last 2 hours. ECG demonstrates ST depression in II, III and aVF, troponin is 0.78 (normal range <0.03). On examination, he is alert but extremely clammy. You note his pulse at the radial artery is 35/ minute and regular. His BP on the cardiac monitor is 120/67. An NSTEMI is diagnosed and the patient is taken to the catheter laboratory for reperfusion therapy. Which coronary artery is most likely to be occluded?

- ○ Left anterior descending artery ×
- ○ Left circumflex artery ×
- ○ Obtuse marginal branch artery 1 ×
- ○ Obtuse marginal branch artery 2 ×
- ○ Right coronary artery ×

Submit answer

Reference ranges

Score: **18.1%**

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Question 73 of 328 ✓

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Left anterior descending artery	3%
Left circumflex artery	3%
Obtuse marginal branch artery 1	2%
Obtuse marginal branch artery 2	0%
Right coronary artery	92%

The sino-atrial node (SAN) is supplied by the right coronary artery in 60-80% of patients, with the left circumflex supplying the remainder of the population. The OM1 and OM2 arteries are branches of the left circumflex artery, classically supplying the apex of the heart.

Discuss (1) | Improve

Next question ›

Coronary circulation

Arterial supply of the heart

- left aortic sinus → left coronary artery (LCA)
- right aortic sinus → right coronary artery (RCA)
- LCA → LAD + circumflex

- RCA → posterior descending
- RCA supplies SA node in 60%, AV node in 90%

Venous drainage of the heart
- coronary sinus drains into the right atrium

The coronary arteries fill during diastole.

123

Next question >

Textbooks

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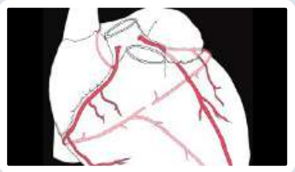
Extended textbook

Media



Coronary circulation of the heart

The Noted Anatomist - YouTube 7 0

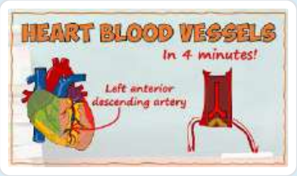
CORONARY CIRCULATION
Osmosis.org

Coronary circulation

Osmosis - YouTube 👍 3 👎 0

Cardiac coronary vessels

Armando Hasudungan - YouTube 👍 1 👎 0


HEART BLOOD VESSELS
In 4 minutes!
Left anterior descending artery

Blood Supply Territories of the Heart

More Than Skin Deep - YouTube 👍 0 👎 0

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Score: **19.2%**

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A 17-year-old man from Romania is seen on the acute medical take. He is usually fit and well but for the past week has had a sore throat and fevers. He was unsure of his vaccination status and a throat swab taken from pustular exudate on his tonsils grew *Corynebacterium diphtheriae* for which he has started erythromycin. His maternal grandfather is known to have glucose-6-phosphate deficiency but he has never received testing.

He presents today with one day of shortness of breath. He is experiencing fevers and having difficulty swallowing. On examination, he is tachypnoeic with a respiratory rate of 34 breaths per minute. He had bibasal crepitations and a JVP can be seen at 4cm above the angle of Louis. He is coughing white frothy material and saturating at 90%.

Na^+	137 mmol/l
K^+	4.2 mmol/l
Urea	6.4 mmol/l
Creatinine	60 µmol/l
Troponin	29,000 ng/ml (normal range <20)
Hb	110 g/l
Platelets	300 * 10^9/l
WBC	9.6 * 10^9/l
Neutrophils	4.3 * 10^9/l
Lymphocytes	8.2 * 10^9/l
Throat swab	*Corynebacterium diphtheriae* resistant to erythromycin, sensitive to penicillin

What complication has arisen?

○ Acute haemolytic crisis ×

○ Myocarditis ×

○ Diphtheria pneumonitis ×

○ Interstitial lung disease ×

○ Lemierre's Syndrome ×

Submit answer

Reference ranges ⌄

Score: **19.2%**

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Urea	6.4 mmol/l
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Neutrophils	4.3 * 10^9/l
Lymphocytes	8.2 * 10^9/l
Throat swab	*Corynebacterium diphtheriae* resistant to erythromycin, sensitive to penicillin

What complication has arisen?

Acute haemolytic crisis 3%

Myocarditis 78%

Diphtheria pneumonitis 5%

Interstitial lung disease 0%

Lemierre's Syndrome 15%

Myocarditis can be a secondary complication of diphtheria

Important for me Less important

The picture shows a large troponin rise with the rapid onset of acute heart failure. Myocarditis is a rare recognised complication of diphtheria and is due to exotoxin produced. The organisms resistance to the macrolide antibiotic has undoubtedly allowed it to persist despite medical therapy. Diphtheria pneumonitis, Lemierre's syndrome and interstitial lung disease could all conceivably present with bibasal crepitations but no troponin rise. An acute haemolytic crisis would usually have a very low haemoglobin and signs of jaundice that are not present here.

👍 👎 Discuss (4) Improve

Next question >

Myocarditis ★

Myocarditis describes inflammation of the myocardium. There are a wide range of underlying causes. It should be particularly considered in younger patients who present with chest pain.

Causes

- viral: coxsackie B, HIV
- bacteria: diphtheria, clostridia
- spirochaetes: Lyme disease
- protozoa: Chagas' disease, toxoplasmosis
- autoimmune
- drugs: doxorubicin

Presentation

- usually young patient with an acute history
- chest pain

- dyspnoea
- arrhythmias

Investigations
- bloods
    - ↑ inflammatory markers in 99%
    - ↑ cardiac enzymes
    - ↑ BNP
- ECG
    - tachycardia
    - arrhythmias
    - ST/T wave changes including ST-segment elevation and T wave inversion

Management
- treatment of underlying cause e.g. antibiotics if bacterial cause
- supportive treatment e.g. of heart failure or arrhythmias

Complications
- heart failure
- arrhythmia, possibly leading to sudden death
- dilated cardiomyopathy: usually a late complication

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

MYOCARDITIS
Osmosis.org

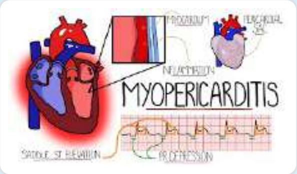
Myocarditis

Osmosis - YouTube 👍 8 👎 0

CARDIOLOGY
MYOCARDITIS

Myocarditis

Armando Hasudungan - YouTube 👍 3 👎 0


MYOCARDIUM
PERICARDIAL
INFLAMMATION
MYOPERICARDITIS
SADDLE ST ELEVATION
PR DEPRESSION

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 0

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Score: **18.9%**

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A 23-year-old gentleman presents with sudden shortness of breath, lethargy and fevers. He is currently homeless and has been brought in by a friend. He has vomited three times since arrival but has no diarrhoea.

He is usually well and has a metal plate in his right wrist for a fracture sustained in an assault a few weeks prior. He does not take any regular medications and is not registered with a local GP. He denies any alcohol or substance use.

He is currently taking rifampicin and isoniazid for pulmonary tuberculosis diagnosed 4 months ago and is compliant with his directly observed therapy.

On examination, he has a pan-systolic murmur over the left sternal edge. He has bilateral lung crepitations to the midzones with dullness to percussion at the base. His heart rate is 96/min, temperature is 37.8ºC and he is saturating at 94% with 4 litres oxygen by nasal cannulae. He has a bounding JVP that reaches the ear lobe with cannon V-waves seen. Tracking marks can be seen in the arms and there are injection sites in his groin. He has poor dentition with tenderness under the left jaw. His right wrist is tender over the plate and there is no redness and a mildly reduced range of motion.

Hb	96 g/l	Na^+	139 mmol/l
Platelets	156 * 10^9/l	K^+	4.2 mmol/l
WBC	14.2 * 10^9/l	Urea	5.6 mmol/l
Neuts	12.0 * 10^9/l	Creatinine	96 µmol/l
CRP	156 mg/l		

Chest x-ray	bilateral alveolar shadowing, small right sided effusion
ECG	T wave inversion in V1-V4
Blood cultures	*Staph aureus*

What is the underlying cause of this presentation?

○ Poor dental hygiene ×

○ Intravenous drug use ×

○ Infected orthopaedic plate ×

○ Tuberculosis ×

○ Prolonged vomiting ×

Submit answer

Reference ranges ∨

Score: **18.9%**

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A 23-year-old gentleman presents with sudden shortness of breath, lethargy and fevers. He is currently homeless and has been brought in by a friend. He has vomited three times since arrival but has no diarrhoea.

He is usually well and has a metal plate in his right wrist for a fracture sustained in an assault a few weeks prior. He does not take any regular medications and is not registered with a local GP. He denies any alcohol or substance use.

He is currently taking rifampicin and isoniazid for pulmonary tuberculosis diagnosed 4 months ago and is compliant with his directly observed therapy.

On examination, he has a pan-systolic murmur over the left sternal edge. He has bilateral lung crepitations to the midzones with dullness to percussion at the base. His heart rate is 96/min, temperature is 37.8ºC and he is saturating at 94% with 4 litres oxygen by nasal cannulae. He has a bounding JVP that reaches the ear lobe with cannon V-waves seen. Tracking marks can be seen in the arms and there are injection sites in his groin. He has poor dentition with tenderness under the left jaw. His right wrist is tender over the plate and there is no redness and a mildly reduced range of motion.

Hb	96 g/l	Na^+	139 mmol/l
Platelets	$156 * 10^9/l$	K^+	4.2 mmol/l
WBC	$14.2 * 10^9/l$	Urea	5.6 mmol/l
Neuts	$12.0 * 10^9/l$	Creatinine	96 µmol/l
CRP	156 mg/l		

Chest x-ray	bilateral alveolar shadowing, small right sided effusion
ECG	T wave inversion in V1-V4
Blood cultures	*Staph aureus*

What is the underlying cause of this presentation?

Poor dental hygiene	5%
Intravenous drug use	84%
Infected orthopaedic plate	10%
Tuberculosis	1%
Prolonged vomiting	0%

Tricuspid valve endocarditis can cause tricuspid regurgitation, which may manifest with a new pan-systolic murmur, large V waves and features of pulmonary emboli

Important for me Less important

This gentleman has infective endocarditis. The cannon V waves and pan systolic murmur narrow this down to the tricuspid valve being affected which is very common in intravenous drug users as this is the first valve reached from venous blood. The tracking marks are a clue even though the patient denies this. Tuberculosis does not cause murmurs and usually only affects the pericardium of the heart. Poor dental hygiene can cause a *strep* endocarditis that is less florid and more insidious. An infected orthopaedic plate is a rare cause of endocarditis. Prolonged vomiting would not cause this. The lack of pitting oedema suggests this is a rapid onset right sided heart failure.

👍 👎 Discuss (4) Improve

Next question >

Tricuspid regurgitation ★

Signs

- pan-systolic murmur
- prominent/giant V waves in JVP
- pulsatile hepatomegaly
- left parasternal heave

Causes

- right ventricular infarction
- pulmonary hypertension e.g. COPD
- rheumatic heart disease
- infective endocarditis (especially intravenous drug users)
- Ebstein's anomaly
- carcinoid syndrome

123

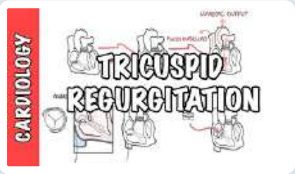
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


CARDIOLOGY
TRICUSPID REGURGITATION

TyszyqQhBfc

Armando Hasudungan - YouTube 👍 1 👎 1

Tricuspid valve disease

Osmosis - YouTube 👍 1 👎 1

Report broken media

Score: **18.7%**

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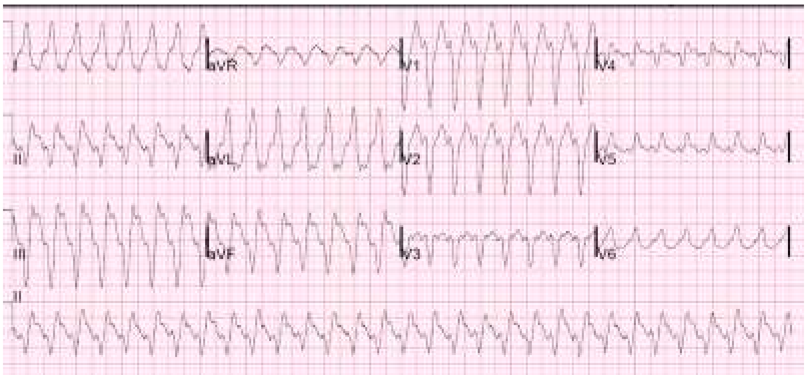
A 68-year-old man attends the emergency department with palpitations and dizziness. This had a sudden onset approximately one hour ago. He denies chest pain. His observations are as follows: heart rate 170 beats per minute, BP 100/70 mmHg, respiratory rate 18 breaths per minute, and temperature 37.2 °C.

An ECG is performed in the department:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

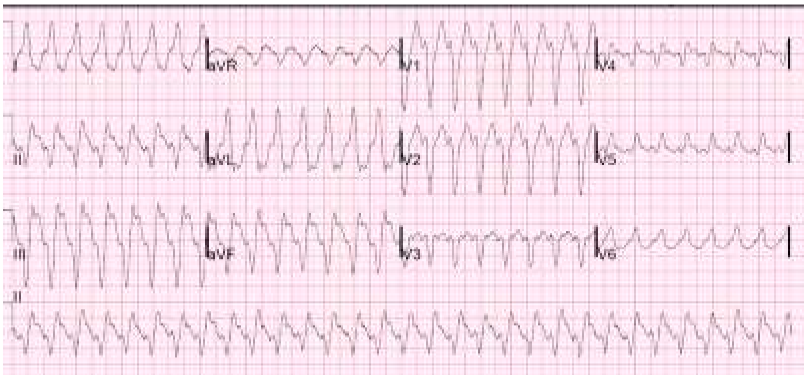
- ○ Atrial fibrillation with aberrant conduction ×
- ○ Monomorphic ventricular tachycardia (VT) ×
- ○ Polymorphic ventricular tachycardia (VT) ×
- ○ Supraventricular tachycardia with aberrant conduction ×
- ○ Ventricular flutter ×

Submit answer

Reference ranges

A 68-year-old man attends the emergency department with palpitations and dizziness. This had a sudden onset approximately one hour ago. He denies chest pain. His observations are as follows: heart rate 170 beats per minute, BP 100/70 mmHg, respiratory rate 18 breaths per minute, and temperature 37.2 ºC.

An ECG is performed in the department:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Atrial fibrillation with aberrant conduction	3%
Monomorphic ventricular tachycardia (VT)	57%
Polymorphic ventricular tachycardia (VT)	9%
Supraventricular tachycardia with aberrant conduction	28%
Ventricular flutter	3%

The ECG demonstrates a broad-complex tachycardia. It can be difficult to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrant conduction (e.g. LBBB). Therefore it is always safest to assume that the arrhythmia is ventricular in origin until proven otherwise.

Electrocardiographic features that increase the likelihood of VT include:

- Absence of typical RBBB or LBBB morphology
- Extreme axis deviation ('northwest axis'): QRS positive in aVR and negative in I and aVF
- Very broad complexes > 160ms
- AV dissociation
- Capture beats
- Fusion beats
- Positive concordance throughout the precordial leads
- Negative concordance throughout the precordial leads
- RSR' complexes with an R>R'

Monomorphic ventricular tachycardia is the correct option. The ECG demonstrates RSR' complexes with an R>R' which is the most specific finding in favour of VT. Since the morphology of the QRS waves is similar beat to beat then the diagnosis is monomorphic VT rather than polymorphic VT.

Atrial fibrillation with aberrant conduction is incorrect. The presence of P waves (seen best in lead II) and the regular rhythm make this an unlikely diagnosis.

Polymorphic ventricular tachycardia is incorrect. This would also present with a broad complex tachycardia with similar criteria as discussed above. However, in this condition, the morphology of the ventricular complex would vary from beat to beat.

Supraventricular tachycardia with aberrant conduction is incorrect. Although this is a possibility, the extremely broad QRS duration and the presence of RSR' complexes with R>R' favour the diagnosis of monomorphic ventricular tachycardia.

Ventricular flutter is incorrect. This condition is an extreme form of ventricular tachycardia (VT) with loss of organised electrical activity. The ECG would demonstrate a continuous sine wave with no identifiable P waves, QRS complexes, or T waves.

👍 👎 💬 Discuss (7) Improve

Next question ›

Peri-arrest rhythms: tachycardia

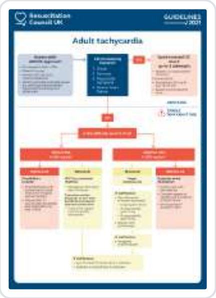
The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given. Up to 3 shocks can be given; after this expert help should be sought.

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment guidelines can be found at the Resuscitation Council website, below is a very limited summary:



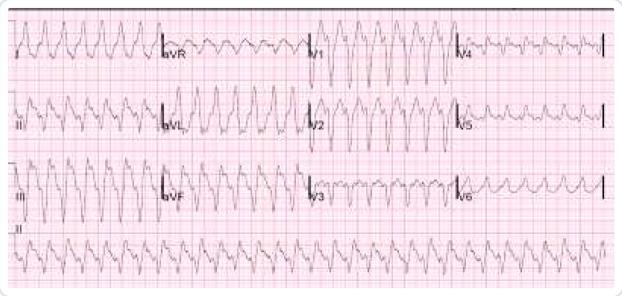
Broad-complex tachycardia

Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion

Irregular

- seek expert help. Possibilities include:
    - atrial fibrillation with bundle branch block - the most likely cause in a stable patient
    - atrial fibrillation with ventricular pre-excitation
    - torsade de pointes



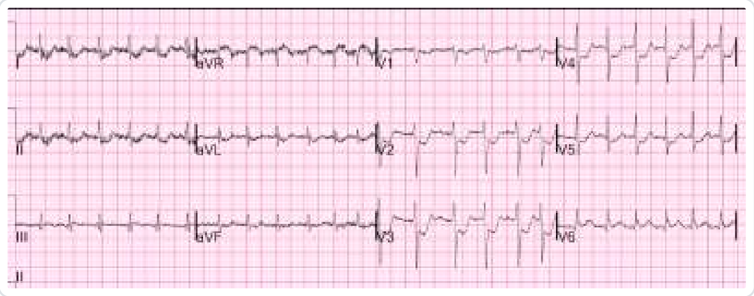
Narrow-complex tachycardia

Regular

- vagal manoeuvres followed by IV adenosine
- if the above is unsuccessful consider a diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control: beta-blockers are usually first-line unless there is a contraindication



+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

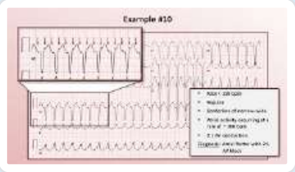
Links

Resuscitation Council UK 👍 30 👎 9

2021 Tachycardia algorithm

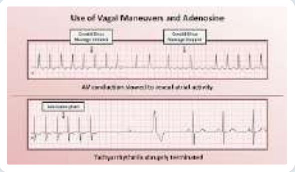
Suggest link Report broken link

Media



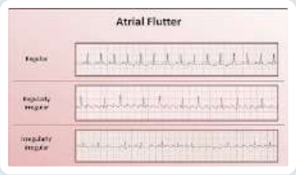
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 5 👎 0


Use of Vagal Maneuvers and Adenosine

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 4 👎 0


Atrial Flutter
Regular
Regularly irregular
Irregularly irregular

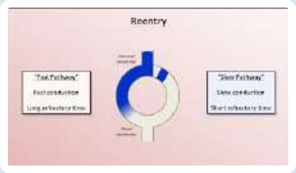
Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 1

CARDIOLOGY
TACHYCARDIA
ARRHYTHMIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 1


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 2 👎 1

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Score: **18.4%**

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Question 77 of 328

A 65-year-old with exertional dyspnoea over the past 3 months is referred to the pulmonary hypertension team. An initial echocardiogram demonstrated 65% ejection fraction, preserved left ventricular function with a pulmonary arterial pressure of 72 mmHg. She undergoes a right and left heart catheter, revealing the following saturations:

Right atrium high	60%
Right atrium mid	89%
Right atrium low	70%
Right ventricle high	70%
Right ventricle mid	73%
Right ventricle low	72%
Pulmonary artery	71%
Capillary wedge	96%

What is the most likely diagnosis?

- ○ Aortic stenosis
- ○ Atrial septal primum defect
- ○ Atrial septal secundum defect
- ○ Pulmonary stenosis
- ○ Mitral stenosis

Submit answer

Reference ranges

Score: **18.4%**

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Question 77 of 328 ✗

A 65-year-old with exertional dyspnoea over the past 3 months is referred to the pulmonary hypertension team. An initial echocardiogram demonstrated 65% ejection fraction, preserved left ventricular function with a pulmonary arterial pressure of 72 mmHg. She undergoes a right and left heart catheter, revealing the following saturations:

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Right atrium mid	89%
Right atrium low	70%
Right ventricle high	70%
Right ventricle mid	73%
Right ventricle low	72%
Pulmonary artery	71%
Capillary wedge	96%

What is the most likely diagnosis?

Aortic stenosis	2%
🟥 Atrial septal primum defect	25%
🟩 Atrial septal secundum defect	60%
Pulmonary stenosis	9%
Mitral stenosis	3%

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation. It is present between the high and mid right atrium, demonstrating the presence of a left-to-right shunt. Primum ASD defects typically occur lowering in the septum than secundum defects, which typically can be found in the mid-atrial region.

👍 | 👎 | Discuss (9) | Improve

Next question >

Cardiac catheterisation and oxygen saturation levels ★

Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usually produce the answer.

Let's start with the basics:

- deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC). It has an oxygen saturation level of around **70%**. The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70%
- the lungs oxygenate the blood to a level of around **98-100%**. The left atrium (LA), left ventricle (LV) and aorta should all, therefore, have oxygen saturation levels of 98-100%

The table below shows the **oxygen saturations** that would be expected in different scenarios:

Diagnosis & notes	**RA**	**RV**	**PA**	**LA**	**LV**	**Aorta**
Normal	70%	70%	70%	100%	100%	100%
Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards	85%	85%	85%	100%	100%	100%

Diagnosis & notes	**RA**	**RV**	**PA**	**LA**	**LV**	**Aorta**
Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated	70%	85%	85%	100%	100%	100%
Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PA having intermediate oxygenation levels	70%	70%	85%	100%	100%	100%
VSD with Eisenmenger's	70%	70%	70%	100%	85%	85%
PDA with Eisenmenger's	70%	70%	70%	100%	100%	85%
ASD with Eisenmenger's	70%	70%	70%	85%	85%	85%

AI + Q 123

Next question ›

Textbooks

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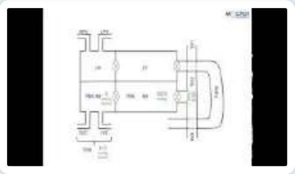
Extended textbook

Media



Interpreting Cardiac Catheterisation Data

MRCPCH Revision - YouTube 👍 25 👎 5

Report broken media

Score: **18.2%**

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Question 78 of 328

A 30-year-old man presents with a fever. He complains that he has had a fever, headache and lethargy for three weeks. On examination, his temperature is 38.5C. You notice a healed human bite mark on his right forearm, he tells you he was bitten in a pub brawl about a couple months ago. Diastolic and systolic murmurs are heard on auscultation. A trans-oesophageal echo demonstrates an oscillating mass on a bicuspid aortic valve, with aortic regurgitation. Three blood cultures are taken before administration of empirical antibiotics, two grow small colonies of tiny pleomorphic gram-negative bacilli.

What is the likely causative organism?

- ○ *Eikenella corrodens* ×
- ○ *Escherichia coli* ×
- ○ *Phoenicoparrus andinus* ×
- ○ *Staphylococcus aureus* ×
- ○ *Streptococcus viridans* ×

Submit answer

Reference ranges

Score: **18.2%**

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A 30-year-old man presents with a fever. He complains that he has had a fever, headache and lethargy for three weeks. On examination, his temperature is 38.5C. You notice a healed human bite mark on his right forearm, he tells you he was bitten in a pub brawl about a couple months ago. Diastolic and systolic murmurs are heard on auscultation. A trans-oesophageal echo demonstrates an oscillating mass on a bicuspid aortic valve, with aortic regurgitation. Three blood cultures are taken before administration of empirical antibiotics, two grow small colonies of tiny pleomorphic gram-negative bacilli.

What is the likely causative organism?

🟩 *Eikenella corrodens*	62%
Escherichia coli	13%
Phoenicoparrus andinus	8%
🟥 *Staphylococcus aureus*	6%
Streptococcus viridans	10%

This patient fits the Duke criteria for infective endocarditis as there is: echocardiographic evidence of an intra-cardiac mass associated with a valve, a fever of >38 and two blood cultures of an endocarditis causing microorganism. Additionally, this patients bicuspid aortic valve is a risk factor for developing endocarditis.

Eikenella corrodens is a gram-negative bacilli, which is a commensal of the human mouth. It is a member of the HACEK group, an acronym for a group of organisms that can cause gram-negative endocarditis (*Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species.) The human bite injury and gram-negative culture make *Eikenella corrodens* the most likely causative organism.

Streptococcus viridans and *Staphylococcus aureus* are commoner causes of infective endocarditis, which are easily cultured and are gram-positive.

Escherichia coli could cause endocarditis and is a gram-negative bacilli, but is not the most likely in this case.

Phoenicoparrus andinus is the binomial nomenclature for the rare Andean Flamingo, which are not known to cause infective endocarditis.

Sources:

Patrick WD, Brown WD, Ian Bowmer M, Sinave CP. Infective endocarditis due to Eikenella corrodens: Case report and review of the literature. The Canadian Journal of Infectious Diseases. 1990;1(4):139-142.

Das M, Badley AD, Cockerill FR, Steckelberg JM, Wilson WR. Infective endocarditis caused by HACEK microorganisms. Annu Rev Med. 1997;48:25-33.

👍 👎 Discuss (9) Improve

Next question ›

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
    - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
    - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis

    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*
    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link Report broken link

Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **17.9%**

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Question 79 of 328

A 30-year-old pregnant woman (24 weeks) is admitted to the acute medical unit with sudden onset shortness of breath and pleuritic chest pain. She has no past medical history other than eczema and this is her first pregnancy.

On examination, auscultation of her chest reveals only a mild wheeze with oxygen saturations of 94% on room air and a respiratory rate of 25/min. Her heart sounds are normal, with a heart rate of 97bpm and blood pressure is 105/60 mmHg. An ECG shows sinus rhythm.

The FY1 doctor on the ward has requested a D-dimer to try and 'speed up the diagnosis', which comes back positive.

What is the next appropriate investigation?

- ○ Ventilation-Perfusion (V/Q) scan ×
- ○ Chest x-ray ×
- ○ Peak flow measurement ×
- ○ CT Pulmonary Angiogram (CTPA) ×
- ○ Ultrasound doppler of the lower legs ×

Submit answer

Reference ranges

Score: **17.9%**

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The FY1 doctor on the ward has requested a D-dimer to try and 'speed up the diagnosis', which comes back positive.

What is the next appropriate investigation?

Ventilation-Perfusion (V/Q) scan	21%
Chest x-ray	52%
Peak flow measurement	2%
CT Pulmonary Angiogram (CTPA)	8%
Ultrasound doppler of the lower legs	19%

It is likely that this woman has had a pulmonary embolism given the acute nature of the shortness of breath and pleuritic chest pain, together with tachycardia and reduced oxygen saturations.

The correct way to investigate a pregnant woman with a possible pulmonary embolism is to first perform a chest x-ray. This is vital to ensure other diagnoses such as pneumonia and pneumothorax are identified

Ultrasound doppler of the lower legs is the next step as this can demonstrate a venous thromboembolism of the lower limb vasculature without radiation. If a thrombus is found, appropriate management with low molecular weight heparin can be initiated.

If the chest x-ray and the ultrasound doppler are normal, a ventilation-perfusion scan should be performed. The presence of a normal chest x-ray can essentially function as the ventilation part of the V/Q scan. This allows for a half-dose radiation V/Q scan.

The radiation dose to the foetus of a V/Q scan is slightly higher than that of a CTPA, however both are well below the threshold of specific harm to the foetus. Please note that a CTPA should only be used in the presence of lung disease including asthma or an abnormal chest x-ray, as the radiation dose to the proliferating maternal breasts may be significant in the development of carcinogenesis later in life.

A d-dimer is not appropriate as it may be raised in pregnancy and may lead to further inappropriate tests based on the positive result alone.

Further reading can be found here:
BMJ Rational Testing: Investigating suspected pulmonary embolism in pregnancy
http://www.bmj.com/content/334/7590/418?sso=

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Next question >

Pregnancy: DVT/PE investigation

Guidelines were updated in 2015 by the Royal College of Obstetricians.

For patients with a suspected deep vein thrombosis (DVT):

- Compression duplex ultrasound should be undertaken where there is clinical suspicion of DVT

For patients with a suspected pulmonary embolism (PE):

- ECG and chest x-ray should be performed in all patients
- In women who also have symptoms and signs of DVT, compression duplex ultrasound should be performed. If compression ultrasonography confirms the presence of DVT, no further investigation is necessary and treatment for VTE should continue

- the decision to perform a V/Q or CTPA should be taken at a local level after discussion with the patient and radiologist

Comparing CTPA to V/Q scanning in pregnancy

CTPA	V/Q scanning
CTPA slightly increases the lifetime risk of **maternal breast cancer** (increased by up to 13.6%, background risk of 1/200 for study population). Pregnancy makes breast tissue particularly sensitive to the effects of radiation	V/Q scanning carries a slightly increased risk of **childhood cancer** compared with CTPA (1/50,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it often raised in pregnancy.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCOG 2 0

Thromboembolic disease in pregnancy and the puerperium: acute management

Suggest link

Report broken link

Score: **19%**

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A 23-year-old Afro-Caribbean female presents with a four-month history of fatigue, transient fever lasting hours, 12kg weight loss and non-specific bilateral headaches. She has presented to the Emergency Department four times in the past 3 months with non-specific abdominal pains that are worse after eating and also blue-lighted into her local hyperacute stroke unit as a thrombolysis patient after sudden onset loss of monocular blindness in her left eye, which resolved before any treatment was given. She has no other past medical history, does not smoke and drinks minimally.

On examination, cranial nerve examination and fundoscopy are both unremarkable. Upper and lower limb neurology are intact with downgoing plantars. Her heart sounds demonstrates a gallop rhythm, an early diastolic murmur and a mild radial-radial delay. Chest auscultation is clear. She appears very warm and measures 38.4ºC in your clinic with facial flushing. Blood tests are as follows:

Hb	94 g/l
Platelets	245 * 10^9/l
WBC	18.4 * 10^9/l
Eosinophil	0.1 * 10^9/l
ESR	121 mm/hr

Na^+	141 mmol/l
K^+	4.0 mmol/l
Urea	5.2 mmol/l
Creatinine	68 µmol/l
CRP	56 mg/l

Complement levels were reported as normal and an antibody screen including ANCA and ANA was negative. Urine dip is negative. Chest X-ray demonstrates focal consolidation. Her heart rate is 95 and regular, her blood pressure is 185/110 mmHg.

What is the unifying diagnosis?

○ Systemic lupus erythematosus ×

○ Polyarteritis nodosa ×

○ Poorly controlled primary hypertension ×

○ Ascending aortic aneurysm ×

○ Takayasu arteritis ×

Submit answer

Reference ranges ⌵

Score: **19%**

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Urea	5.2 mmol/l
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CRP	56 mg/l

Complement levels were reported as normal and an antibody screen including ANCA and ANA was negative. Urine dip is negative. Chest X-ray demonstrates focal consolidation. Her heart rate is 95 and regular, her blood pressure is 185/110 mmHg.

What is the unifying diagnosis?

Systemic lupus erythematosus	3%
Polyarteritis nodosa	21%
Poorly controlled primary hypertension	1%
Ascending aortic aneurysm	5%
Takayasu arteritis	70%

A systemic condition is clearly present in this patient: there is a combination of fever, weight loss, pyrexia, ischaemic abdominal pain, transiently ischaemic cerebral occlusion, raised inflammatory markers and hypertension associated with radial-radial delay. The key differentials are between PAN and Takayasu arteries, which are medium-sized and large vessel vasculitis respectively. Ischaemic colitis is a common presentation in PAN but almost always associated with one of positive ANCA, eosinophilia, peripheral neuropathy and hepatitis B. In addition, the involvement of temporal arteries and it appears the ascending aorta to produce radial-radial delay suggests Takayasu arteritis to be the most likely diagnosis. Immediate CT or MRI imaging is necessary with acute immunosuppression with high-dose steroids, followed by maintenance immunosuppression with azathioprine, methotrexate or cyclophosphamide.

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Takayasu's arteritis ★

Takayasu's arteritis is a large vessel vasculitis. It typically causes occlusion of the aorta and questions commonly refer to an absent limb pulse. It is more common in younger females (e.g. 10-40 years) and Asian people.

Features

- systemic features of a vasculitis e.g. malaise, headache
- unequal blood pressure in the upper limbs
- carotid bruit and tenderness
- absent or weak peripheral pulses
- upper and lower limb claudication on exertion
- aortic regurgitation (around 20%)

Associations

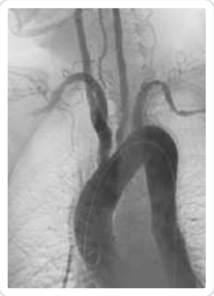
- renal artery stenosis

Investigations

- vascular imaging of the arterial tree is required to make a diagnosis of Takayasu's arteritis
  - either magnetic resonance angiography (MRA) or CT angiography (CTA)

Management

- steroids



123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

[Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology]

Armando Hasudungan - YouTube 👍 4 👎 0

Takayasu arteritis
Khan Academy

Takayasu arteritis

Khan Academy - YouTube 👍 4 👎 0

Report broken media

Score: **18.8%**

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A 73-year-old man was admitted as an emergency suffering from severe, central and crushing chest pain. ECG revealed evidence of anterior ST elevation MI and primary percutaneous coronary intervention was performed with the deployment of two drug-eluting stents to the left anterior descending artery. The patient had no previous coronary artery disease or significant family history. He is an ex-smoker and drinks approximately 10 units of alcohol per week.

Post-procedure the patient was well and mobilised on the ward without further chest pain or shortness of breath. Clinical examination was unremarkable.

The patient was commenced on aspirin, clopidogrel, ramipril, bisoprolol and atorvastatin therapy. Further investigations performed following intervention are summarised as below.

Haemoglobin	13.9 d / dL
White cell count	8.6 * 10^9/l
Platelets	199 * 10^9/l
Urea	7.2 mmol / L
Creatinine	110 micromol / L
Sodium	137 mmol / L
Potassium	4.3 mmol / L
Total cholesterol	5.4 mmol / L
LDL cholesterol	3.1 mmol / L
HDL cholesterol	1.2 mmol / L
Triglycerides	2.5 mmol / L

Transthoracic echocardiogram: no valvular abnormality; mild anterior dyskinesia with overall normal left ventricular systolic function; ejection fraction 55-60 %

Primary PCI report: total occlusion of mid-LAD, two DES deployed with good angiographic result; 50 % mid-vessel occlusion of the right coronary artery; circumflex unobstructed.

Prior to discharge, the patient asks to speak to you about any other possible strategies to reduce his risk of suffering a further heart attack.

Which of the following interventions is recommended for this patient by NICE guidance on secondary prevention of myocardial infarction?

○ Percutaneous coronary intervention to right coronary artery ×
○ Mediterranean-style diet ×
○ Abstinence from alcohol ×
○ Omega-3 fatty acid supplements ×
○ Aldosterone antagonist (for example, eplerenone) ×

Submit answer

Reference ranges ⌄

Score: **18.8%**

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LDL cholesterol	3.1 mmol / L
HDL cholesterol	1.2 mmol / L
Triglycerides	2.5 mmol / L

Transthoracic echocardiogram: no valvular abnormality; mild anterior dyskinesia with overall normal left ventricular systolic function; ejection fraction 55-60 %

Primary PCI report: total occlusion of mid-LAD, two DES deployed with good angiographic result; 50 % mid-vessel occlusion of the right coronary artery; circumflex unobstructed.

Prior to discharge, the patient asks to speak to you about any other possible strategies to reduce his risk of suffering a further heart attack.

Which of the following interventions is recommended for this patient by NICE guidance on secondary prevention of myocardial infarction?

Percutaneous coronary intervention to right coronary artery	9%
Mediterranean-style diet	52%
Abstinence from alcohol	20%
Omega-3 fatty acid supplements	7%
Aldosterone antagonist (for example, eplerenone)	12%

NICE recommends several lifestyle changes after a myocardial infarction. These include smoking cessation, a Mediterranean-style diet, achievement of a healthy weight, limitation of alcohol consumption to recommended healthy limits and 20-30 minutes of physical activity daily. NICE does not recommend routinely eating oily fish or the consumption of omega-3 supplements, although it is noted that there is no evidence of harm from these interventions.

Aldosterone antagonists are recommended for use in patients with symptoms and signs of heart failure or with evidence of systolic impairment on echocardiography. There is no evidence that percutaneous treatment of mild asymptomatic coronary artery disease (such as in this patient) reduces the future risk of myocardial infarction.

Jones K, Saxon L, Cunningham W, Adams P. Secondary prevention for patients after a myocardial infarction: summary of updated NICE guidance. BMJ 2013;347:f6544.

👍 👎 Discuss (8) Improve

Next question ›

Myocardial infarction: secondary prevention ★

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2013. Some key points are listed below

All patients should be offered the following drugs:

- dual antiplatelet therapy (aspirin plus a second antiplatelet agent)
- ACE inhibitor
- beta-blocker
- statin

Some selected lifestyle points:

- diet: advise a Mediterranean style diet, switch butter and cheese for plant oil based products. Do not recommend omega-3 supplements or eating oily fish
- exercise: advise 20-30 mins a day until patients are 'slightly breathless'
- sexual activity may resume 4 weeks after an uncomplicated MI. Reassure patients that sex does not increase their likelihood of a further MI. PDE5 inhibitors (e.g, sildenafil) may be used 6 months after a MI. They should however be avoided in patient prescribed either nitrates or nicorandil

Most patients who've had an acute coronary syndrome are now given dual antiplatelet therapy (DAPT). Clopidogrel was previously the second antiplatelet of choice. Now ticagrelor and prasugrel (also ADP-receptor inhibitors) are more widely used. NICE now recommend:

- post acute coronary syndrome (medically managed): add ticagrelor to aspirin, stop ticagrelor after 12 months
- post percutaneous coronary intervention: add prasugrel or ticagrelor to aspirin, stop the second antiplatelet after 12 months
- this 12 month period may be altered for people at a high-risk of bleeding or those who at high-risk of further ischaemic events

Aldosterone antagonists

- patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment (e.g. eplerenone) should be initiated within 3-14 days of the MI, preferably after ACE inhibitor therapy

+ 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 6 👎 8

2017 STEMI guidelines

Clinical Knowledge Summaries 👍 8 👎 5

Antiplatelet guidelines

NICE 👍 16 👎 5

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Score: **18.5%**

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A 69-year-old man with a background of ischaemic heart disease attends his routine cardiology appointment. He reports a 2-month history of progressively worsening shortness of breath and is only able to walk 100 yards before needing to stop for rest. He has occasional exertional chest pain which resolves with sublingual glyceryl trinitrate. He is currently taking aspirin, atorvastatin, lansoprazole, bisoprolol, ramipril, spironolactone and sublingual glyceryl trinitrate as required.

On examination, his heart rate is 80bpm and regular with a blood pressure of 130/75mmHg. His chest is clear with good air entry bilaterally and saturations of 94% on room air. He has a soft and non-tender abdomen with no palpable organomegaly. There is pitting oedema to his knees.

ECG: Sinus rhythm, Q waves and T wave inversion in V1-V4, QRS duration 110ms.

Echocardiogram: Left ventricular ejection fraction 33%, pulmonary arterial pressure 20mmHg.

What is the most appropriate step in the management of this patient?

- ○ Cardiac resynchronisation therapy
- ○ Digoxin
- ○ Furosemide
- ○ Ivabradine
- ○ Nicorandil

Submit answer

Reference ranges

Score: **18.5%**

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A 69-year-old man with a background of ischaemic heart disease attends his routine cardiology appointment. He reports a 2-month history of progressively worsening shortness of breath and is only able to walk 100 yards before needing to stop for rest. He has occasional exertional chest pain which resolves with sublingual glyceryl trinitrate. He is currently taking aspirin, atorvastatin, lansoprazole, bisoprolol, ramipril, spironolactone and sublingual glyceryl trinitrate as required.

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ECG: Sinus rhythm, Q waves and T wave inversion in V1-V4, QRS duration 110ms.

Echocardiogram: Left ventricular ejection fraction 33%, pulmonary arterial pressure 20mmHg.

What is the most appropriate step in the management of this patient?

Cardiac resynchronisation therapy	15%
Digoxin	3%
Furosemide	16%
Ivabradine	62%
Nicorandil	5%

Ivabradine should be considered in heart failure if the patient has sinus rhythm > 75/min and a LVEF < 35% and have not responded to to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Important for me Less important

Ivabradine is a 3rd line agent in the treatment of heart failure. It works by selectively inhibiting the 'funny current' I*f*, also known as the pacemaker current found in the sinoatrial node. The drug acts to lower the heart rate and therefore the oxygen demand needed to supply to the myocardium. Criteria for prescribing ivabradine include a heart rate > 75bpm and an ejection fraction of < 35%.

Cardiac resynchronisation therapy (CRT) is another 3rd line treatment agent for heart failure. It is indicated for patients with left ventricular dysfunction and consequently a reduced LV stroke volume which is complicated by a dyssynchronous ventricular contraction secondary to a conduction delay (usually LBBB). It involves inserting electrodes into the walls of the ventricle to coordinate their contraction and ensure a synchronised contraction of the left ventricle to increase the ejection fraction. An indication for its use is a reduced ejection fraction (< 35%) and widened QRS complexes (>120ms) on the ECG.

Digoxin can be used as a 3rd line agent in the treatment of heart failure. It is strongly indicated in patients with co-existing atrial fibrillation.

Furosemide can provide symptomatic relief from fluid overload in heart failure. However, it does not improve mortality and is therefore not the next recommended treatment option.

Nicorandil is a potassium channel agonist that causes vasodilatation and is therefore used in the treatment of stable angina. Due to the effects of vasodilation on blood pressure and ejection fraction, its use is contraindicated in those with left ventricular dysfunction.

👍 👎 Discuss (7) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

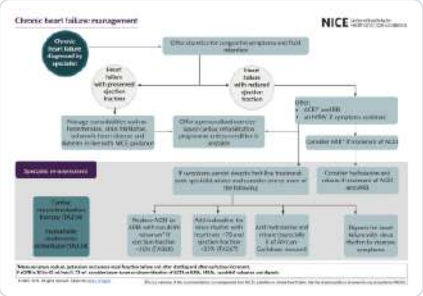
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine

    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction
Specialist treatment

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

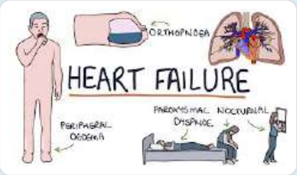
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

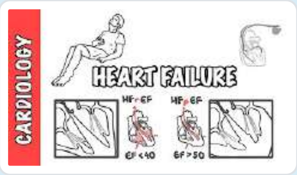
Suggest link Report broken link

Media


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
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EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

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Score: **18.3%**

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Question 83 of 328

A 29-year-old man is admitted to the cardiology ward following a collapse whilst playing football. He has no recollection of events other than running on the pitch and waking up in the ambulance. In the last 2-3 months, he has been complaining of intermittent palpitations but put this down to work-related stress. There is no history of chest pain and he has no past medical history of note.

His observations are recorded within normal limits. On auscultation, chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen and his neurological examination is unremarkable.

A bedside echocardiogram demonstrates a hypokinetic right ventricle.

Given the likely diagnosis, what is the most characteristic finding on this patient's ECG?

- ○ Left ventricular hypertrophy ×
- ○ Positive deflection at the J point ×
- ○ Right bundle brach block ×
- ○ Slurred upstroke in QRS ×
- ○ Small positive deflection at the end of QRS complex ×

Submit answer

Reference ranges

Score: **18.3%**

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A 29-year-old man is admitted to the cardiology ward following a collapse whilst playing football. He has no recollection of events other than running on the pitch and waking up in the ambulance. In the last 2-3 months, he has been complaining of intermittent palpitations but put this down to work-related stress. There is no history of chest pain and he has no past medical history of note.

His observations are recorded within normal limits. On auscultation, chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen and his neurological examination is unremarkable.

A bedside echocardiogram demonstrates a hypokinetic right ventricle.

Given the likely diagnosis, what is the most characteristic finding on this patient's ECG?

Left ventricular hypertrophy	11%
Positive deflection at the J point	8%
Right bundle brach block	20%
Slurred upstroke in QRS	13%
Small positive deflection at the end of QRS complex	48%

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

Important for me Less important

This patient has arrhythmogenic right ventricular dysplasia (ARVD), a type of inherited cardiovascular disease. ARVD can present as palpitations, syncope, or sudden cardiac death similar to hypertrophic obstructive cardiomyopathy (HOCM). Over time, the right ventricular myocardium is replaced by fibrofatty tissue and patients develop signs of right ventricular failure. This patient's echocardiogram finding of a hypokinetic right ventricular wall is suggestive of the diagnosis. The most characteristic ECG finding in ARVD is an **epsilon wave**. An epsilon wave is a positive deflection following the end of the QRS complex caused by post-excitation of right ventricular myocytes.

Left ventricular hypertrophy is a common ECG finding of HOCM. HOCM is the most common cause of sudden cardiac death and can also present with palpitations and syncope, as seen in this patient. However, this patient's echocardiogram findings are in keeping with ARVD, of which the epsilon wave is the most characteristic ECG finding.

An **Osborn wave (or J wave) is a positive deflection at the J point**. There are multiple causes, of which the most common is hypothermia. Other causes of J waves include left ventricular hypertrophy and Brugada syndrome. However, an Osborn wave is not associated with ARVD.

There are multiple causes of **right bundle branch block**, including right ventricular hypertrophy. In ARVD, rather than hypertrophy of the right ventricle, the myocardium is replaced by fatty tissue. Right bundle branch block is not a sign of ARVD. Instead, the left bundle branch block is more likely to feature on an ECG of a patient with ARVD.

A **delta wave** is a slurred upstroke in the QRS complex that is commonly associated with pre-excitatory activity, such as that seen in Wolff-Parkinson White syndrome. It is not associated with ARVD.

👍 👎 Discuss (3) Improve

Next question >

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology

- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation

- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 13 👎 7

Arrhythmogenic right ventricular cardiomyopathy ECG

Suggest link

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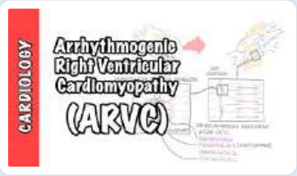
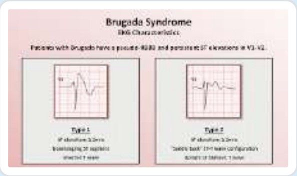
Media


CARDIOLOGY
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome
EKG Characteristics

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

Report broken media

Score: **19.3%**

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Question 84 of 328

A medical opinion was sought from the obstetrics team regarding a 38-year-old 28 weeks pregnant lady. A routine blood pressure check revealed a blood pressure of 158/98 mmHg. Other than suffering from hyperemesis gravidarum, her pregnancy had proceeded without complication. She specifically denied the presence of headaches, vomiting, change in vision, abdominal pain, seizures or bleeding per vagina. She had noticed no change in the frequency of foetal movements, and her 20-week antenatal scan revealed the presence of a healthy foetus with a rate of growth within the expected range. Her past medical history was unremarkable; she was a non-smoker and did not consume alcohol. Her blood pressure at the booking antenatal appointment was 148/88 mmHg. Her sister suffered from pre-eclampsia during her pregnancy necessitating delivery by caesarean section.

Examination revealed the presence of a well 28-week old pregnant lady. Her blood pressure was indeed 158/98 mmHg, her heart rate was 86 bpm and temperature 36.5ºC. Examination of the cardiovascular system revealed normal heart sounds, a JVP of 3cm and the absence of pedal oedema. Examination of the respiratory system was unremarkable. Examination of the gastrointestinal system revealed the presence of a symphysiofundal height appropriate for the stage of gestation with easily obtainable foetal heart sounds on hand held Doppler examination. Examination of the neurological system was unremarkable with normal reflexes, cranial nerve function and peripheral motor and sensory function. Urinalysis revealed no abnormality.

What is the next best management step?

- ○ Commence ramipril ×
- ○ Commence labetalol ×
- ○ Commence indapamide ×
- ○ Commence magnesium sulphate ×
- ○ Transfer to high dependency unit to observe for signs of pre

eclampsia

Submit answer

Reference ranges

Score: **19.3%**

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What is the next best management step?

Commence ramipril	1%
Commence labetalol	90%
Commence indapamide	1%
Commence magnesium sulphate	3%

Transfer to high dependency unit to observe for signs of pre eclampsia 5%

This lady has pre-existing hypertension; her blood pressure at the antenatal booking clinic was elevated and continues to be elevated throughout (as opposed to gestational hypertension in which hypertension develops after 20 weeks). If untreated hypertension is associated with adverse maternal and foetal outcomes including intrauterine growth restriction, placental abruption, cerebrovascular accidents and prematurity. There are no clinical features of preeclampsia, including notably the presence of proteinuria and peripheral oedema, and there is, therefore, no indication for admission to a high dependency unit or to commence magnesium sulphate. Of the remaining options, labetalol is the safest antihypertensive to use in pregnancy; methyldopa is an alternative. The usual first line ACE inhibitors are absolutely contraindicated in pregnancy.

👍 👎 Discuss (7) Improve

Next question >

Pre-eclampsia ★

Pre-eclampsia describes the emergence of high blood pressure during pregnancy that may be a precursor to a woman developing eclampsia and other complications. It is classically a triad of 3 things:

- new-onset hypertension
- proteinuria
- oedema

Definition

The current formal definition is as follows

- new-onset blood pressure ≥ 140/90 mmHg after 20 weeks of pregnancy, AND 1 or more of the following:
    - proteinuria
    - other organ involvement (see list below for examples): e.g. renal insufficiency (creatinine ≥ 90 umol/L), liver, neurological, haematological, uteroplacental dysfunction

Features

Potential consequences of pre-eclampsia

- eclampsia
    - other neurological complications include altered mental status, blindness, stroke, clonus, severe headaches or persistent visual scotomata
- fetal complications
    - intrauterine growth retardation
    - prematurity
- liver involvement (elevated transaminases)
- haemorrhage: placental abruption, intra-abdominal, intra-cerebral
- cardiac failure

Features of severe pre-eclampsia

- hypertension: typically > 160/110 mmHg and proteinuria as above
- proteinuria: dipstick ++/+++
- headache
- visual disturbance
- papilloedema
- RUQ/epigastric pain
- hyperreflexia
- platelet count < 100 * 10^6/l, abnormal liver enzymes or HELLP syndrome

Prevention

NICE divide risk factors into high and moderate risk:

High risk factors	Moderate risk factors
• hypertensive disease in a previous pregnancy • chronic kidney disease • autoimmune disease, such as systemic lupus erythematosus or antiphospholipid syndrome • type 1 or type 2 diabetes • chronic hypertension	• first pregnancy • age 40 years or older • pregnancy interval of more than 10 years • body mass index (BMI) of 35 kg/m² or more at first visit • family history of pre-eclampsia

High risk factors	Moderate risk factors
	- multiple pregnancy

Reducing the risk of hypertensive disorders in pregnancy

- women with the following should take aspirin 75-150mg daily from 12 weeks gestation until the birth
  - ≥ 1 high risk factors
  - ≥ 2 moderate factors

Management

Initial assessment

- NICE recommend arranging emergency secondary care assessment for any woman in whom pre-eclampsia is suspected
- women with blood pressure ≥ 160/110 mmHg are likely to be admitted and observed

Further management

- oral labetalol is now first-line following the 2010 NICE guidelines. Nifedipine (e.g. if asthmatic) and hydralazine may also be used
- delivery of the baby is the most important and definitive management step. The timing depends on the individual clinical scenario

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 20 👎 4

2019 Hypertension in pregnancy: diagnosis and management

Suggest link Report broken link

Media

Preeclampsia & eclampsia

Osmosis - YouTube 👍 1 👎 0

Pre Eclampsia - Overview

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **19%**

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Question 85 of 328

A 38-year-old female of Asian descent, with no significant past medical history, presents after a syncopal event while pruning hedges. She has had a 6-month history of fever, arthralgia and for the past few weeks has had multiple episodes of vertigo and one syncopal event. She denies headaches or visual complaints. Her examination reveals a diminished radial pulse in the left arm and a systolic blood pressure difference in the upper extremities of 14 mmHg. A bruit is auscultated along the left upper extremity. Dopplers of the upper extremities indicate a stenotic area along the subclavian that is later confirmed by Magnetic Resonance Angiography (MRA). She is diagnosed with subclavian steal syndrome. Laboratory tests reveal a normocytic normochromic anaemia, elevated CRP and ESR, negative ANA and ANCA, and all other laboratory tests are within normal range. Of the following, What is the most likely diagnosis?

- ○ Fibromuscular dysplasia ×
- ○ Ehlers-Danlos syndrome ×
- ○ Takayasu arteritis ×
- ○ Giant cell arteritis ×
- ○ Wegener's granulomatosis ×

Submit answer

Reference ranges

Score: **19%**

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Fibromuscular dysplasia	7%
Ehlers-Danlos syndrome	2%
Takayasu arteritis	88%
Giant cell arteritis	3%
Wegener's granulomatosis	1%

This patient likely has Takayasu arteritis which is essentially a chronic vasculitis primarily of the aorta and its branches. These patients can present in a variety of different ways depending on the vessels affected, however, they all typically have a prodrome of systemic symptoms including fatigue, weight loss and low-grade fevers prior to developing any vascular complaints. From a pathology standpoint biopsies of vessels are very similar to giant cell and are typically not performed.

There are 6 criteria for the diagnosis of Takayasu. Presence of 3 of the 6 has 90% sensitivity and specificity for diagnosis:

- 1. Age onset <=40 years
- 2. Claudication of the extremities
- 3. Decreased pulsation of one or both brachial arteries

- 4. Difference of at least 10 mm Hg in systolic blood pressure between the arms
- 5. Bruit over one or both subclavian arteries or the abdominal aorta
- 6. Arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or other causes

Fibromuscular dysplasia typically affects the renal arteries leading to renal artery stenosis and hypertension, and it not accompanied by other systemic manifestations like fever and malaise. Ehlers-Danlos syndrome is a genetic defect in type III collagen and can lead to aneurysms along with hyperelasticity of the skin and hypermobile joints, but other systemic manifestations are typically not present. Giant cell arteritis is most similar to Takayasu in pathology, however typically affects older patients and usually presents with headaches and tenderness over the temporal artery. Lastly, Wegener's is actually a small vessel vasculitis and its most common presenting symptoms include persistent rhinorrhoea, purulent/bloody nasal discharge, oral and/or nasal ulcers, polyarthralgias, myalgias, or sinus pain. Most with Wegener's are ANCA positive.

https://www.rheumatology.org/practice/clinical/classification/takayasu.asp

👍 | 👎 | Discuss (5) | Improve

Next question >

Takayasu's arteritis ★

Takayasu's arteritis is a large vessel vasculitis. It typically causes occlusion of the aorta and questions commonly refer to an absent limb pulse. It is more common in younger females (e.g. 10-40 years) and Asian people.

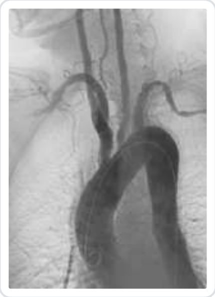
Features
- systemic features of a vasculitis e.g. malaise, headache
- unequal blood pressure in the upper limbs
- carotid bruit and tenderness
- absent or weak peripheral pulses
- upper and lower limb claudication on exertion

- aortic regurgitation (around 20%)

Associations
- renal artery stenosis

Investigations
- vascular imaging of the arterial tree is required to make a diagnosis of Takayasu's arteritis
    - either magnetic resonance angiography (MRA) or CT angiography (CTA)

Management
- steroids



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Next question

Textbooks

High-yield textbook

Extended textbook

Media

Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology

Armando Hasudungan - YouTube 👍 4 👎 0

Takayasu arteritis
Khan Academy

Takayasu arteritis

Khan Academy - YouTube 👍 4 👎 0

Report broken media

Score: **18.8%**

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A 72-year-old woman with a 30 year history of type 2 diabetes mellitus comes for review. She was diagnosed with chronic kidney disease (secondary to diabetes) 8 years ago and has seen declining renal function since. Her current medication includes ramipril 10mg od, amlodipine 10mg od, simvastatin 40mg on and Novomix 30 insulin bd.

Her most recent renal function tests show the following:

Na^+	139 mmol/l
K^+	5.3 mmol/l
Urea	10.2 mmol/l
Creatinine	123 µmol/l
eGFR	40 ml/min/1.73m²

Blood pressure in clinic is 156/88 mmHg and this is confirmed on a second reading. What should be done regarding her blood pressure medication?

- ○ Add bisoprolol
- ○ Add indapamide
- ○ Add doxazosin
- ○ Add spironolactone
- ○ Add an angiotensin II receptor blocker

Submit answer

Reference ranges

Score: **18.8%**

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Urea	10.2 mmol/l
Creatinine	123 µmol/l
eGFR	40 ml/min/1.73m²

Blood pressure in clinic is 156/88 mmHg and this is confirmed on a second reading. What should be done regarding her blood pressure medication?

Add bisoprolol	9%
Add indapamide	72%
Add doxazosin	13%
Add spironolactone	3%
Add an angiotensin II receptor blocker	3%

Poorly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

Important for me Less important

Tight blood pressure remains a key management aim in patients with diabetic nephropathy. ACE inhibitors are clearly the most evidence based management in this arena but her blood pressure is persistently high. If we look at the NICE guidelines the next step would be the addition of a thiazide based diuretic (e.g. indapamide) and there is no reason not to follow these recommendations in this situation.

If the eGFR is less than 30 ml/min/1.73m^2 then thiazides should be avoided as the BNF states:

> *Thiazides and related diuretics are ineffective if eGFR is less than 30 mL/minute/1.73 m2 and should be avoided; metolazone remains effective but with a risk of excessive diuresis*

Spironolactone and angiotensin II receptor blockers may risk precipitating hyperkalaemia.

👍 👎 Discuss (5) Improve

Next question ›

Hypertension: management ★

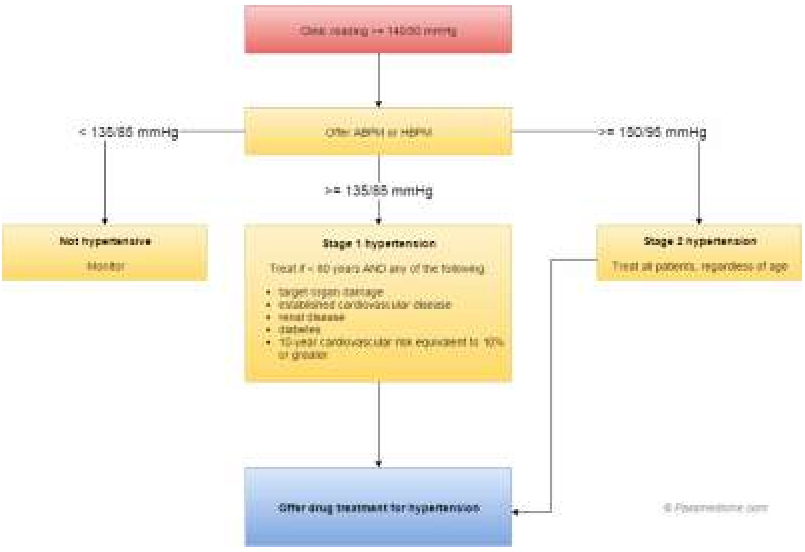
NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	**Criteria**
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

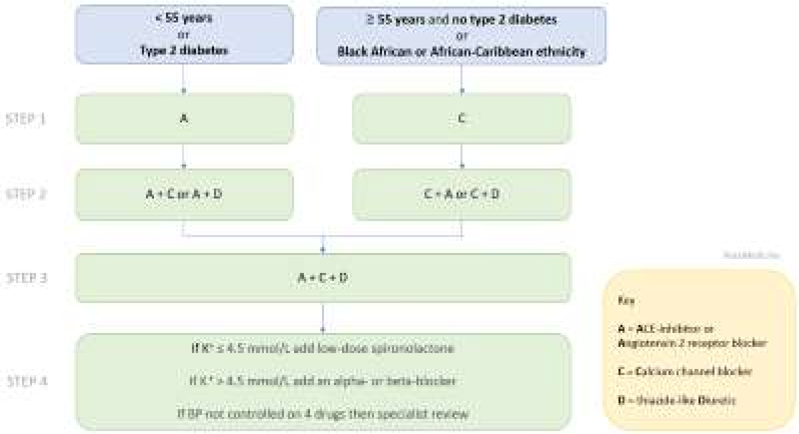
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
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C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**

  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
  - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

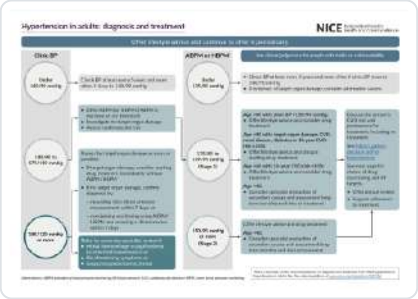
- add a third drug to make, i.e.:
  - if already taking an **(A + C)** then add a **D**
  - if already **(A + D)** then add a **C**
- **(A + C + D)**

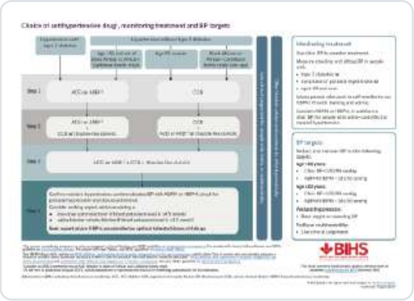
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
  - confirm elevated clinic BP with ABPM or HBPM
  - assess for postural hypotension.
  - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*





Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

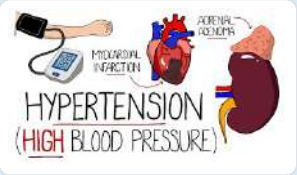
Links

NICE 👍 162 👎 162

[2019 Hypertension guidelines]

Suggest link Report broken link

Media


MYOCARDIAL INFARCTION
ADRENAL ADENOMA
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **18.6%**

1 ✗
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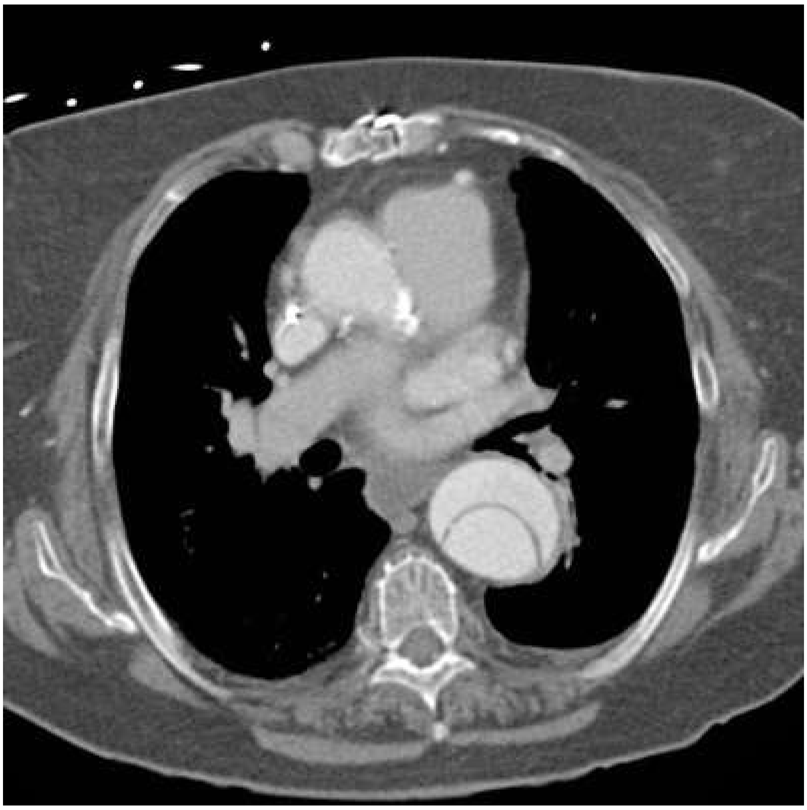
A 74-year-old man with a history of depression, chronic obstructive pulmonary disease and hypertension is admitted to hospital with severe central chest pain. He has no history of ischaemic heart disease or similar chest pains in the past. On admission blood pressure is 160/98 mmHg, pulse 110/min, respiratory rate 18/min and oxygen saturations are 93% on room air.

A CT chest (with contrast) is shown below:



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What is the most appropriate management?

○ Chest drain

○ Cardiothoracic surgery

○ Intravenous labetalol

○ Thrombolysis with tenecteplase

○ Low-molecular weight heparin ×

Submit answer

Reference ranges ⌵

Score: **18.6%**

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Question 87 of 328 ✗

A 74-year-old man with a history of depression, chronic obstructive pulmonary disease and hypertension is admitted to hospital with severe central chest pain. He has no history of ischaemic heart disease or similar chest pains in the past. On admission blood pressure is 160/98 mmHg, pulse 110/min, respiratory rate 18/min and oxygen saturations are 93% on room air.

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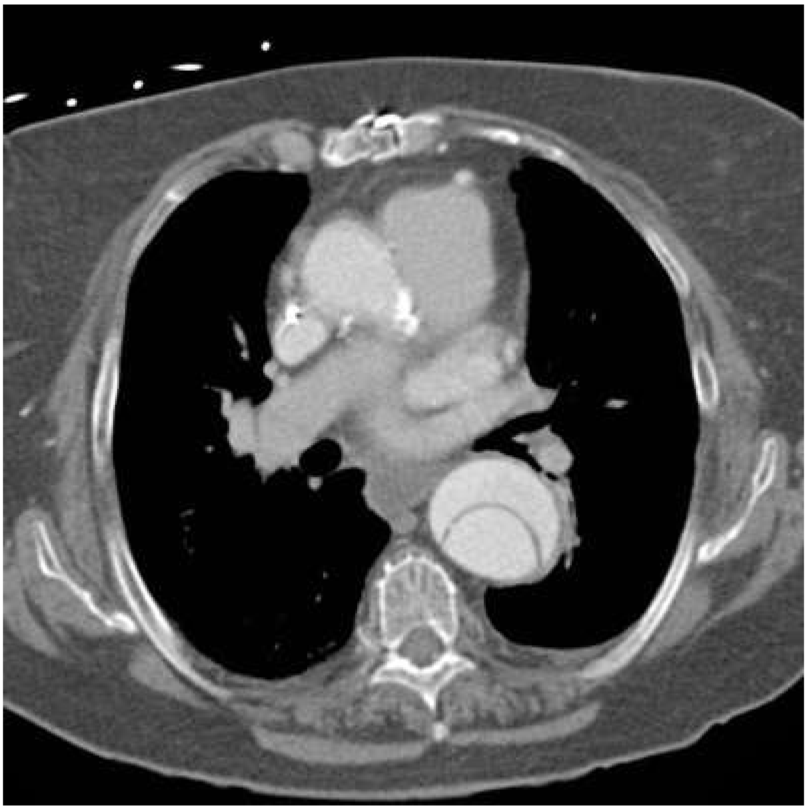

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What is the most appropriate management?

Chest drain	1%
Cardiothoracic surgery	21%
Intravenous labetalol	69%
Thrombolysis with tenecteplase	4%

Low-molecular weight heparin 5%

Dissection of the descending aorta can be seen. As this is a Stanford type B dissection it is treated conservatively with intravenous beta-blockers to lower blood pressure and help prevent any extension.

Discuss (7) Improve

Next question >

Aortic dissection: investigation and management

Classification

Stanford classification

- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally

Investigation

It's important to remember that patients may present acutely and be clinically unstable. The choice of investigations will have to take account of this.

Chest x-ray

- widened mediastinum

CT angiography of the chest, abdomen and pelvis is the investigation of choice

- suitable for stable patients and for planning surgery
- a false lumen is a key finding in diagnosing aortic dissection

Transoesophageal echocardiography (TOE)

- more suitable for unstable patients who are too risky to take to CT scanner

Management

Type A

- surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention

Type B*

- conservative management
- bed rest
- reduce blood pressure IV labetalol to prevent progression

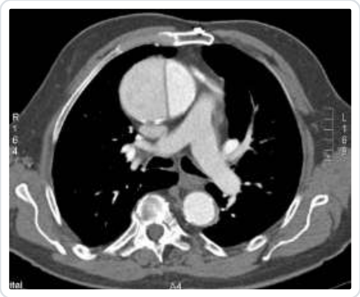
Complications

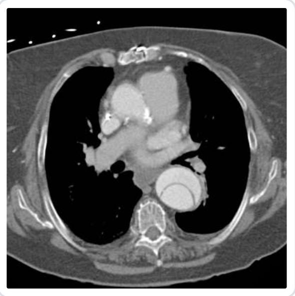
Complications of backward tear

- aortic incompetence/regurgitation
- MI: inferior pattern is often seen due to right coronary involvement

Complications of a forward tear

- unequal arm pulses and BP
- stroke
- renal failure





*endovascular repair of type B aortic dissection may have a role in the future

123

Next question >

Textbooks

High-yield textbook

Extended textbook

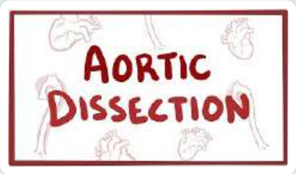
Links

American Heart Association 13 13

2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease

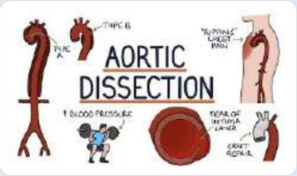
Suggest link Report broken link

Media



Aortic dissection

Osmosis - YouTube 👍 9 👎 0


AORTIC DISSECTION
TYPE B
TYPE A
RIPPING CHEST PAIN
↑ BLOOD PRESSURE
TEAR OF INTIMA LAYER
GRAFT REPAIR

Understanding Aortic Dissection

Zero To Finals - YouTube 👍 1 👎 0

Report broken media

Score: **18.4%**

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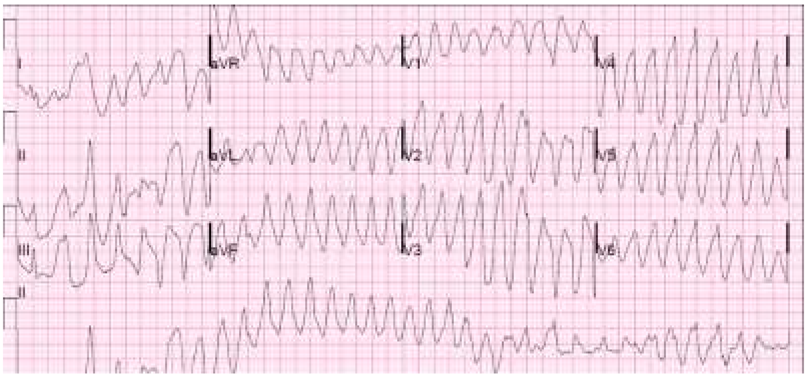
Question 88 of 328

A 44-year-old woman presents to the emergency department with dyspnoea. She has been feeling intermittently dizzy and short-of-breath for the past 2 weeks. On examination her pulse is 180/min, blood pressure 100/66 mmHg, oxygen saturations 98% on room air. Her chest is clear and she appears well perfused. An ECG is obtained:



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What is the most appropriate treatment? ,

○ Intravenous amiodarone ×

○ Intravenous adenosine ×

○ Intravenous magnesium ×

○ Intravenous labetalol ×

○ Unsynchronised DC shock ×

Submit answer

Reference ranges

Score: **18.4%**

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Back to top

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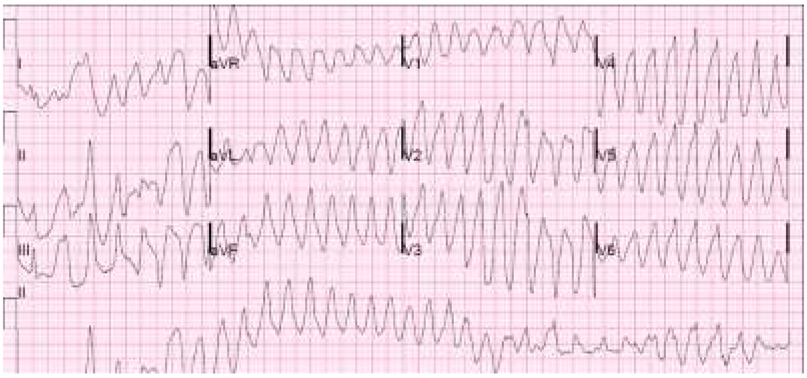

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What is the most appropriate treatment? ,

Intravenous amiodarone	14%
Intravenous adenosine	1%
Intravenous magnesium	80%
Intravenous labetalol	0%
Unsynchronised DC shock	5%

IV magnesium sulfate is used to treat torsades de pointes

Important for me Less important

The ECG shows an irregular wide-complex tachycardia, a form of polymorphic ventricular tachycardia or more specifically torsades de pointes. This patient had an underlying long QT interval secondary to a combination of medications. The acute treatment for this is intravenous magnesium.

Precipitating medications should, of course, be reviewed and electrolyte abnormalities corrected.

If the patient was in shock or periarrest then the ALS tachycardia should be followed, i.e. SYNCHRONISED DC shocks. There are, however, no 'adverse' signs in this patient.

The ALS periarrest guidelines state the following:

> *Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Do not give amiodarone for definite torsade de pointes. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulfate 2 g IV over 10 min (= 8 mmol, 4 mL of 50% magnesium sulfate).*

👍 👎 Discuss (3) Improve

Next question >

Torsades de pointes ★

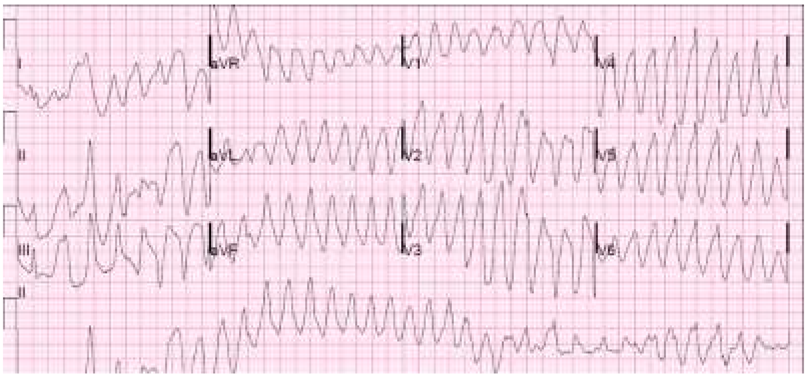
Torsades de pointes ('twisting of the points') is a form of polymorphic ventricular tachycardia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death.

Causes of long QT interval
- congenital
  - Jervell-Lange-Nielsen syndrome
  - Romano-Ward syndrome
- antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- tricyclic antidepressants
- antipsychotics
- chloroquine

- terfenadine
- erythromycin
- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- myocarditis
- hypothermia
- subarachnoid haemorrhage

Management

- IV magnesium sulphate



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ECG showing torsades de pointes

+ Q 123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 19 👎 3

Torsades de pointes

Suggest link Report broken link

Media

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **18.2%**

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A 67-year-old woman is complaining of a six-hour history of chest pain and dizziness in the high-dependency unit. She underwent a mitral valve replacement five days ago and had some temporary trans-venous pacing wires removed earlier today.

On examination, she has oxygen saturations of 93% on 2 litres via nasal cannula, has a heart rate of 110/min, a blood pressure of 76/43mmHg, has a temperature of 37.9ºC, and is responsive to voice. She feels cool peripherally. An ECG is performed at the bedside which shows sinus rhythm with QRS complexes of alternating amplitude.

Blood tests from this morning show the following:

Hb	89 g/L	(115 - 160)
Platelets	121 * 10^9/L	(150 - 400)
WBC	13.1 * 10^9/L	(4.0 - 11.0)
Na^+	143 mmol/L	(135 - 145)
K^+	3.1 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	151 µmol/L	(55 - 120)
CRP	282 mg/L	(< 5)
Magnesium	0.83 mmol/L	(0.85 - 1.10)

What is the most appropriate management given the likely diagnosis?

○ Electrolyte replacement

○ Emergency percutaneous coronary intervention

○ Pericardiocentesis

○ Prolonged course of antibiotics

○ Urgent temporary pacing wire insertion

Submit answer

Reference ranges ⌄

Score: **18.2%**

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89	-

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WBC	13.1 * 10^9/L	(4.0 - 11.0)
Na^+	143 mmol/L	(135 - 145)
K^+	3.1 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	151 µmol/L	(55 - 120)
CRP	282 mg/L	(< 5)
Magnesium	0.83 mmol/L	(0.85 - 1.10)

What is the most appropriate management given the likely diagnosis?

Electrolyte replacement	10%
Emergency percutaneous coronary intervention	2%
Pericardiocentesis	76%
Prolonged course of antibiotics	5%
Urgent temporary pacing wire insertion	7%

Electrical alternans is suggestive of cardiac tamponade

Important for me Less important

This woman has clinical signs of cardiac tamponade which is confirmed by electrical alternans on ECG. Electrical alternans occurs due to the movement or 'swinging' of the heart within the pericardium as a result of the fluid present. This results in a variable amplitude of the QRS complex depending on the hearts proximity to the ECG leads. In this case, this was likely precipitated by a traumatic removal of trans-venous pacing wires causing bleeding into the pericardial space. As a result, she requires an urgent echocardiogram to confirm the diagnosis and subsequent **pericardiocentesis** or re-sternotomy to drain the blood. It is worth noting that her acute kidney injury is the result of poor cardiac output as a result of her tamponade.

Although there are electrolyte abnormalities on this patient's blood tests that should be replaced, this is not likely to be the cause of her acute presentation. An arrhythmia post-operatively could cause a reduction in cardiac output however the ECG performed here demonstrates sinus rhythm and therefore **electrolyte replacement** would not reverse her current condition.

An acute coronary syndrome must be considered in cardiac post-operative patients complaining of chest pain however the electrical alternans on ECG with no remarkable ST changes to speak of is more consistent with a diagnosis of cardiac tamponade. Therefore, **emergency percutaneous coronary intervention** is not the most appropriate option here.

A **prolonged course of antibiotics** may be employed if a diagnosis of infective endocarditis is suspected however this is not the case here for a variety of reasons. Firstly, infective endocarditis post-operatively is unlikely to present within 5 days. Furthermore, the electrical alternans on ECG is indicative of cardiac tamponade rather than infective endocarditis. Finally, although the patient has a fever and a raised CRP, these are common findings post-operatively due to the systemic inflammatory response to surgery and are not specific for infective endocarditis.

Arrhythmias are common in the post-operative period for patients who have undergone cardiac surgery, particularly valve surgery, however, this patient is in sinus rhythm at an acceptable rate. Therefore, as this patient's pathology is mechanical rather than electrical, **urgent temporary pacing wire insertion** will not help with her current issue.

👍 👎 Discuss (3) Improve

Next question >

Cardiac tamponade ★

Cardiac tamponade is characterized by the accumulation of pericardial fluid under pressure.

Classical features - Beck's triad:

- hypotension
- raised JVP
- muffled heart sounds

Other features:

- dyspnoea
- tachycardia
- an absent Y descent on the JVP - this is due to the limited right ventricular filling
- pulsus paradoxus - an abnormally large drop in BP during inspiration
- Kussmaul's sign - much debate about this
- ECG: electrical alternans

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

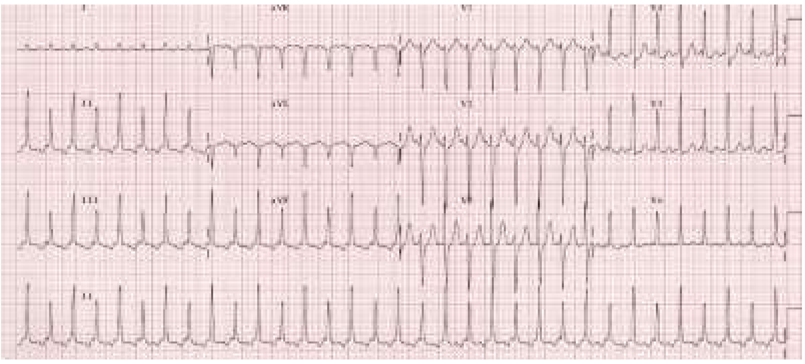
	Cardiac tamponade	**Constrictive pericarditis**
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present

	Cardiac tamponade	Constrictive pericarditis
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Management

- urgent pericardiocentesis



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An ECG demonstrating electrical alternans. Note the alternation of QRS complex amplitude between beats.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 12 👎 4

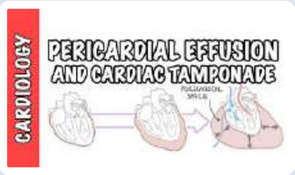
ECG Findings in Massive Pericardial Effusion

Suggest link Report broken link

Media

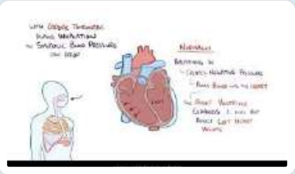
Echocardiogram of a Patient With Cardiac Tamponade

JAMA - YouTube 👍 12 👎 0


CARDIOLOGY
PERICARDIAL EFFUSION
AND CARDIAC TAMPONADE

Cardiac tamponade

Armando Hasudungan - YouTube 👍 1 👎 0



Cardiac tamponade

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **18%**

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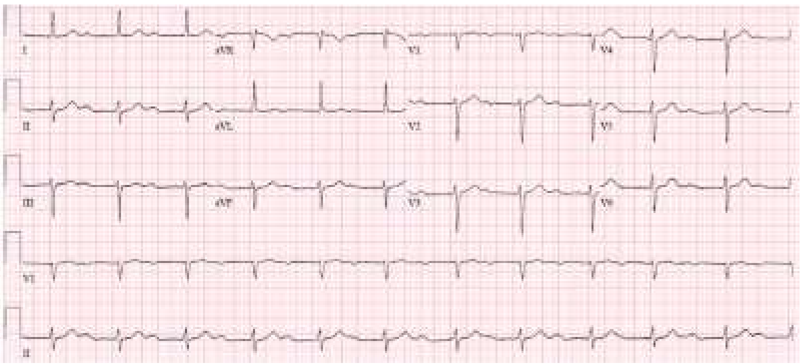
Question 90 of 328

A 58-year-old man is referred to the cardiology clinic for a review. He complains of a 4-week history of intermittent palpitations and chest discomfort for approximately 1-2 minutes. He works as a stockbroker and has been recently promoted. There is no past medical history of note and he is an active tennis player on the weekends. He takes no regular medications and drinks 1 bottle of wine per week.

On examination, he has a heart rate of 65 beats/min and is regular with a blood pressure of 131/68mmHg. He has normal heart sounds and equal air entry on auscultation. His calves are soft with no pitting oedema.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the recommended management for this patient?

- ○ Catheter ablation of accessory pathway ×
- ○ Oral aspirin ×
- ○ Oral bisoprolol ×
- ○ Reassurance and discharge ×
- ○ Referral for pacemaker insertion ×

Submit answer

A 58-year-old man is referred to the cardiology clinic for a review. He complains of a 4-week history of intermittent palpitations and chest discomfort for approximately 1-2 minutes. He works as a stockbroker and has been recently promoted. There is no past medical history of note and he is an active tennis player on the weekends. He takes no regular medications and drinks 1 bottle of wine per week.

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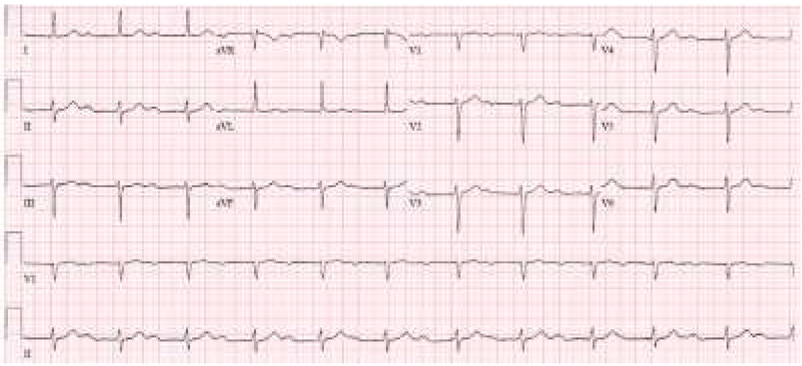


© Image used on license from Dr Smith, University of Minnesota

What is the recommended management for this patient?

- Catheter ablation of accessory pathway 14%
- Oral aspirin 1%
- Oral bisoprolol 4%
- Reassurance and discharge 52%
- Referral for pacemaker insertion 29%

Reassurance and discharge is correct. This patient's ECG shows first-degree heart block with a prolonged PR interval. First-degree heart block is not of any clinical concern and does not warrant further management. Taking a closer look at this patient's presentation, it seems that his chest discomfort and palpitations have coincided with a new job promotion and could be related to stress or anxiety. This history is atypical for cardiac ischaemia and there are minimal risk factors present to suggest that this patient's presentation was related to sinister cardiac pathology.

Catheter ablation of accessory pathway is incorrect. An accessory pathway (e.g. Bundle of Kent) is capable of creating a re-entry circuit that predisposes the patient to tachyarrhythmias. An example of a re-entry tachycardia associated with an accessory pathway is Wolff-Parkinson-White (WPW) syndrome. ECG features of WPW include PR-interval shortening, QRS prolongation and the classic 'delta wave' (the slow, slurred uprising of the QRS complex). These features are not seen on this patient's ECG. Catheter ablation is also used in the treatment of atrial fibrillation, but this patient has a regular rhythm with visible p waves, making atrial fibrillation very unlikely.

Oral aspirin is incorrect. This is used in the management of ischaemic heart disease that appears as the presence of Q waves, ST-segment elevation or depression or T wave inversion. None of these symptoms are seen, making ischaemic heart disease unlikely and, therefore, the use of aspirin incorrect.

Oral bisoprolol is incorrect. This is used in the management of ectopic burden and tachyarrhythmias. However, it is not used in the management of first-degree heart block.

Referral for pacemaker insertion is incorrect. Indications for pacemaker insertion include complete heart block, second-degree mobitz type 2 heart block, ventricular pauses >3 seconds and previous asystole. This patient has first-degree heart block which is not an indication to be referred for permanent pacemaker insertion.

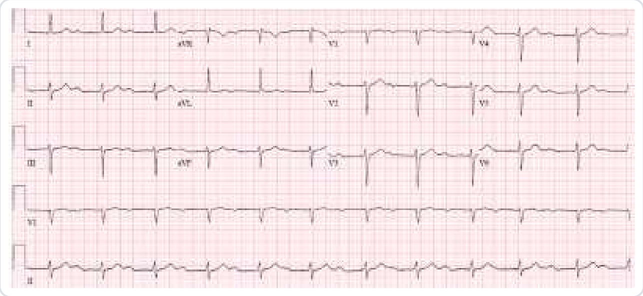
👍 | 👎 | Discuss (6) | Improve

Next question >

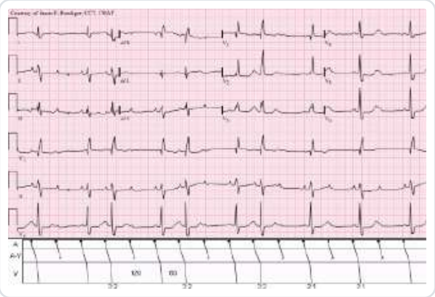
Atrioventricular block ★

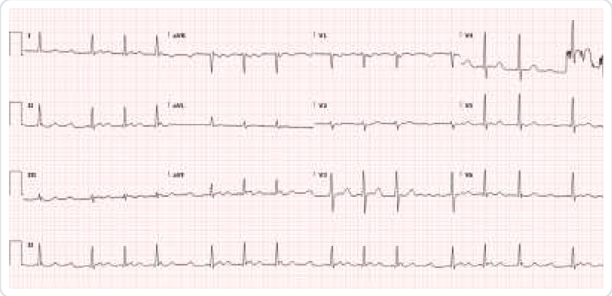
In atrioventricular (AV) block, or heart block, there is impaired electrical conduction between the atria and ventricles. There are three types:

First-degree heart block
- PR interval > 0.2 seconds
- asymptomatic first-degree heart block is relatively common and does not need treatment

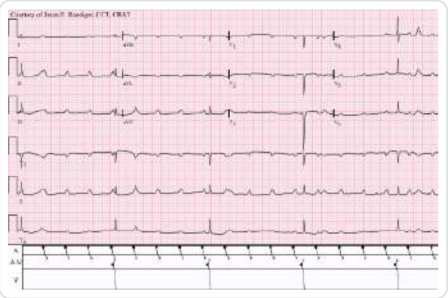


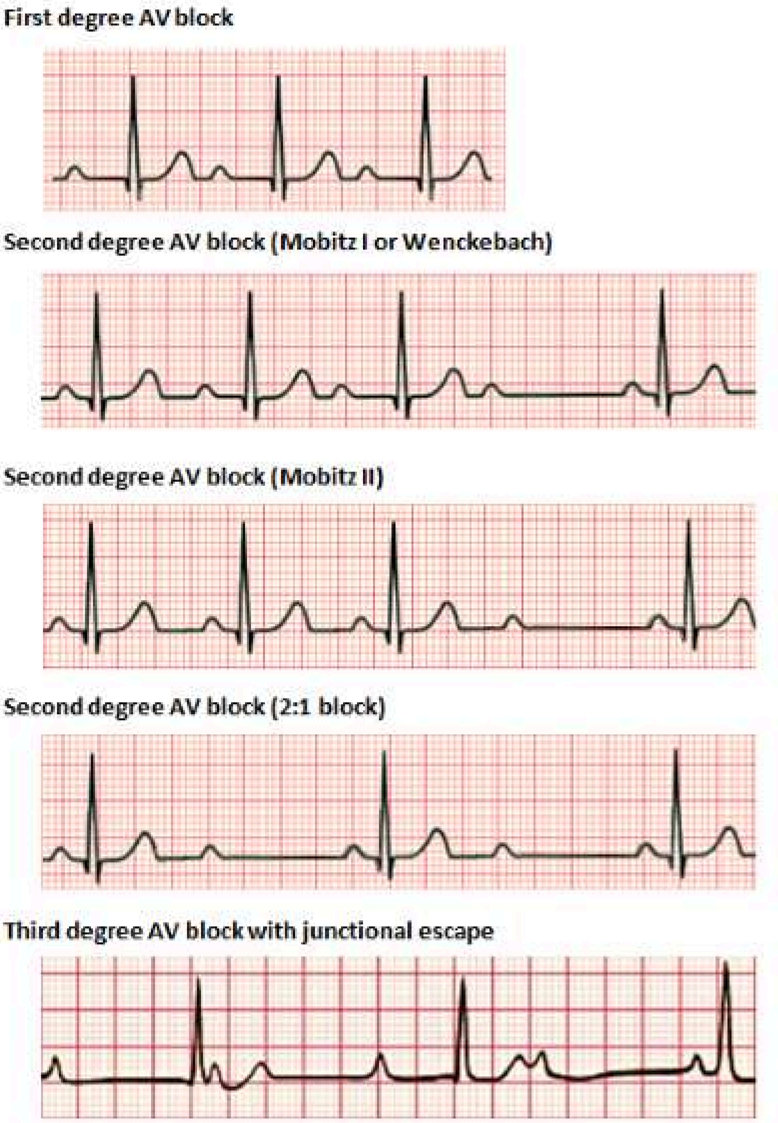
Second-degree heart block
- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex





Third-degree (complete) heart block
- there is no association between the P waves and QRS complexes




First degree AV block
Second degree AV block (Mobitz I or Wenckebach)
Second degree AV block (Mobitz II)
Second degree AV block (2:1 block)
Third degree AV block with junctional escape

ECG showing types of heart block

123

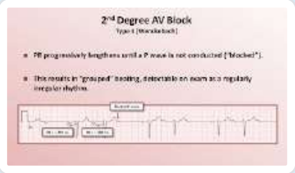
Next question

Textbooks

High-yield textbook

Extended textbook

Media

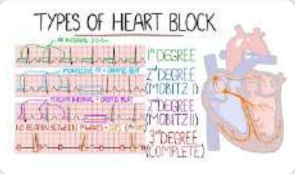

2nd Degree AV Block

AV block

Strong Medicine - YouTube 👍 7 👎 0

Atrioventricular block

Osmosis - YouTube 👍 1 👎 0


TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **17.8%**

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Question 91 of 328

An 83-year-old male presents with ischaemic sounding chest pain that has persisted for the past one hour. A 12-lead ECG is performed and shows deep T wave inversion in leads V1 and V2.

Which is the most likely implicated coronary artery?

- ○ Left circumflex artery
- ○ Left main stem artery
- ○ Proximal left anterior descending artery
- ○ Right coronary artery
- ○ Distal left anterior descending artery

Submit answer

Reference ranges

Score: **17.8%**

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Question 91 of 328 ✗

An 83-year-old male presents with ischaemic sounding chest pain that has persisted for the past one hour. A 12-lead ECG is performed and shows deep T wave inversion in leads V1 and V2.

Which is the most likely implicated coronary artery?

Left circumflex artery	7%
Left main stem artery	6%
Proximal left anterior descending artery	62%
Right coronary artery	8%
Distal left anterior descending artery	16%

Ischaemic changes in leads V1-V4 - left anterior descending

Important for me Less important

Wellens' syndrome is an ECG manifestation of critical proximal left anterior descending (LAD) coronary artery stenosis in patients with unstable angina. It is characterized by symmetrical, often deep (>2 mm), T wave inversions in the anterior precordial leads.

Discuss (4) Improve

Next question ›

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	**Coronary artery**
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

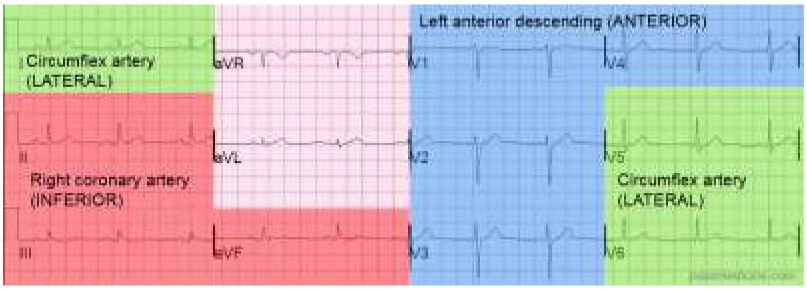
Left anterior descending (ANTERIOR)
Circumflex artery (LATERAL)
aVR
V1
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 24 👎 40

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 14 👎 23

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **17.6%**

1 ✗
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A 22-year-old woman presents to the emergency department with a 1-day history of abdominal pain and vomiting. She denies fever or diarrhoea and disclosed that she went out to a party the night before and consumed 7 glasses of wine. She reports that there is a heart condition that runs in her family, but she cannot remember the name of the condition.

Her observations are within normal limits. On examination, there is central and epigastric tenderness but no guarding or peritonism. Her respiratory and cardiovascular examinations were unremarkable.

Venous blood gas:

pH	7.35	(7.35 - 7.45)
Na^+	138 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Lactate	3.5 mmol/L	(< 1.6)
Glucose	6.2 mmol/L	(4 - 8)

ECG: Sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

What drug is it most important to avoid?

○ Codeine

○ Cyclizine

○ Hyoscine butylbromide

○ Omeprazole

○ Ondansetron

Submit answer

Reference ranges ⌄

Score: **17.6%**

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Question 92 of 328 ✓

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Lactate	3.5 mmol/L	(< 1.6)
Glucose	6.2 mmol/L	(4 - 8)

ECG: Sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

What drug is it most important to avoid?

Codeine	2%
Cyclizine	11%
Hyoscine butylbromide	3%
Omeprazole	4%
Ondansetron	80%

5HT-3 receptor antagonists shouldn't be used as antiemetics in patients with long-QT syndrome

Important for me Less important

This patient has a prolonged QTc interval of 550ms. A normal QTc interval for women should lie between 350-460ms. Patients with a prolonged QTc interval are at particular risk of developing critical arrhythmias such as Torsades de pointes (a polymorphic ventricular tachycardia). It is therefore extremely important to avoid drugs that may prolong the QTc interval further. **Ondansetron** is a type of 5HT-3 receptor antagonist that is associated with QTc interval prolongation and should therefore be avoided.

Codeine is a weak opioid analgesic with common side effects of nausea, drowsiness and constipation. It does not prolong the QTc interval.

Cyclizine is a H1-receptor antagonist. Side effects of cyclizine include urinary retention, dry mouth, and blurred vision. Prolongation of the QTc interval is not a common feature of this medication.

Hyoscine butylbromide is used in the treatment of gastric spasm. It is an anticholinergic agent and is, therefore, contraindicated in myasthenia gravis, urinary retention and gastrointestinal obstruction. It does not prolong the QTc interval.

Omeprazole is a proton-pump inhibitor used in the treatment of gastritis and gastro-oesophageal bleeding. Complications include electrolyte disturbances (e.g. hypomagnesaemia) and increased risk of *Clostridium dificile* infections but not prolongation of the QTc interval.

Discuss (2) Improve

Next question

Long QT syndrome

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1

& LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) • methadone • chloroquine • terfenadine** • erythromycin • haloperidol • ondanestron	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Features
- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management
- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

2010 Drug-Induced Long QT syndrome

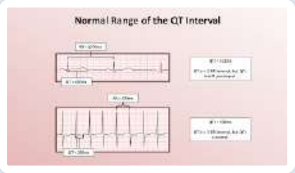
Life in the Fast Lane 👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link

Report broken link

Media


Normal Range of the QT Interval

Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **18.5%**

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Question 93 of 328

A 64-year-old man presents to his general practitioner (GP) with several months of chest pain that occurs only on exertion. When he pauses to rest, the pain dissipates within a few minutes. He has a past medical history of high cholesterol, for which he takes atorvastatin. The GP commences him on bisoprolol.

Several weeks later, the patient re-attends, stating that the symptoms are still not under control.

What should be added next?

- ○ Amlodipine
- ○ Isosorbide mononitrate
- ○ Ivabradine
- ○ Nicorandil
- ○ Verapamil

Submit answer

Reference ranges

Score: **18.5%**

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A 64-year-old man presents to his general practitioner (GP) with several months of chest pain that occurs only on exertion. When he pauses to rest, the pain dissipates within a few minutes. He has a past medical history of high cholesterol, for which he takes atorvastatin. The GP commences him on bisoprolol.

Several weeks later, the patient re-attends, stating that the symptoms are still not under control.

What should be added next?

Amlodipine	69%
Isosorbide mononitrate	18%
Ivabradine	2%
Nicorandil	3%
Verapamil	7%

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

Important for me Less important

This patient is experiencing stable angina - the chest pain is occurring on exertion, but not at rest. NICE recommend the use of a beta-blocker or calcium channel blocker (CCB) first-line. As this patient has been commenced on bisoprolol already, the next step is to add a CCB. A dihydropyridine CCB should be used, to avoid reducing the heart rate further, and so **amlodipine** is the most appropriate option here.

Isosorbide mononitrate would be appropriate to use as a third agent, if the combination of a beta-blocker and CCB was not adequate. However, at this stage, a CCB should be added.

Similarly, **ivabradine**, which modulates the 'funny current', would be appropriate as a third agent instead of isosorbide mononitrate. A CCB should be added first, as this patient is only on bisoprolol.

Nicorandil is another alternative agent to be added third, instead of isosorbide mononitrate or ivabradine. It would not be appropriate to add currently, as the patient is only on bisoprolol.

Verapamil is a non-dihydropyridine CCB. As such, it is negatively chronotropic and so should not be used alongside a beta-blocker; this may precipitate complete heart block. It would have been suitable as a first-line alternative to the beta-blocker.

👍 👎 Discuss (1) Improve

Next question >

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)

- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

Suggest link

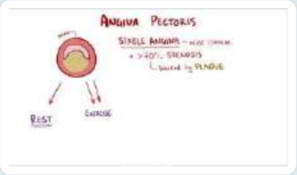
Report broken link

Media

STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

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Question 94 of 328

A 64-year-old man presents to his general practitioner 4 weeks after a permanent pacemaker (PPM) insertion. This was inserted for symptomatic first-degree heart block. He reports that since this was inserted, he has actually felt worse and his exercise tolerance has become more limited.

An ECG is performed and shows a paced rhythm on VVI at 70/min. Each pacing spike is followed by a QRS complex and there are no pacing spikes in between beats. He has regular p waves at a rate of 35/min which are not related to the QRS complexes.

What is the most appropriate intervention?

- ○ Convert to a dual chamber pacemaker ×
- ○ Increase the pacing output ×
- ○ Increase the pacing sensitivity ×
- ○ Reduce the pacing output ×
- ○ Reduce the pacing sensitivity ×

Submit answer

Reference ranges

Score: **18.3%**

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Question 94 of 328 ✓

A 64-year-old man presents to his general practitioner 4 weeks after a permanent pacemaker (PPM) insertion. This was inserted for symptomatic first-degree heart block. He reports that since this was inserted, he has actually felt worse and his exercise tolerance has become more limited.

An ECG is performed and shows a paced rhythm on VVI at 70/min. Each pacing spike is followed by a QRS complex and there are no pacing spikes in between beats. He has regular p waves at a rate of 35/min which are not related to the QRS complexes.

What is the most appropriate intervention?

Convert to a dual chamber pacemaker	60%
Increase the pacing output	11%
Increase the pacing sensitivity	16%
Reduce the pacing output	7%
Reduce the pacing sensitivity	7%

A VVI pacemaker will pace and sense the right ventricle. In the presence or organised atrial activity, a VVI pacemaker may pace the ventricles out of sync with the atria resulting in pacemaker syndrome.

Important for me Less important

This man currently has single-chamber pacing to the ventricles as identified by his mode (VVI). Although this guarantees a ventricular rate, this is at the expense of atrioventricular concordance. For some people, cardiac output may actually fall because of this reduced diastolic function (known as pacemaker syndrome). Hence the worsening of this man's symptoms. One way to correct this would be to **convert to a dual-chamber pacemaker** and establish DDD pacing. This would pace both the atria and ventricles and ensure atrioventricular concordance. Atrial pacing would be possible given that the patient has p waves and therefore is not in atrial fibrillation.

Increasing the pacing output is a useful intervention when there is insufficient capture (the pacing spikes are not at a sufficient voltage to elicit a corresponding QRS complex). As this man's ECG shows good capture, this is not the most appropriate intervention.

An **increase of pacing sensitivity** increases the voltage required to inhibit the pacemaker. This would be a helpful intervention if the pacemaker was being inappropriately inhibited (potentially by noise). As the pacemaker is currently delivering regular pacing spikes at an appropriate rate, over-inhibition is not the cause of this man's symptoms and so this intervention would not be appropriate.

A **reduction in the pacing output** can be helpful to preserve the pacemaker's battery life and therefore the lowest output required to safely and reliably achieve capture should be used at all times, however this will not help with this patient's complaint.

Reducing the pacing sensitivity reduces the voltage required to inhibit the pacemaker. This could be employed if the patient's native rhythm was faster than the rate of the pacemaker but was still not inhibiting the pacemaker from firing. This is particularly important to avoid R on T pacing. As the pacing spikes here are immediately followed by QRS complexes, this man is reliant on his pacemaker's rate and there is no evidence that native QRS complexes are not able to inhibit the pacemaker. Therefore this intervention is not indicated.

👍 👎 Discuss (3) Improve

Next question >

Pacemaker: permanent ★

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:

- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):

- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):

- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):

- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):

- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):

- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:

- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:

- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

DUAL CHAMBER
PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

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Score: **19.1%**

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Question 95 of 328

A 58-year-old female reports having symptomatic episodes of palpitations for the last six months. She is normally fit and drinks the occasional small glass of red wine when she goes out for a meal. The last episode was four days ago when she was cooking in her kitchen. She describes that she felt light headed with central chest discomfort that eased after twenty minutes. In clinic, her observations are all stable and an initial twelve lead electrocardiogram shows sinus rhythm.

What would be the investigation of choice?

- ○ 24 hour ambulatory electrocardiogram ×
- ○ Electrocardiogram treadmill test ×
- ○ 24 hour blood pressure ×
- ○ 72 hour ambulatory electrocardiogram ×
- ○ Event recorder electrocardiogram ×

Submit answer

Reference ranges

Score: **19.1%**

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Question 95 of 328 ✕

A 58-year-old female reports having symptomatic episodes of palpitations for the last six months. She is normally fit and drinks the occasional small glass of red wine when she goes out for a meal. The last episode was four days ago when she was cooking in her kitchen. She describes that she felt light headed with central chest discomfort that eased after twenty minutes. In clinic, her observations are all stable and an initial twelve lead electrocardiogram shows sinus rhythm.

What would be the investigation of choice?

24 hour ambulatory electrocardiogram	21%
Electrocardiogram treadmill test	3%
24 hour blood pressure	1%
72 hour ambulatory electrocardiogram	42%
Event recorder electrocardiogram	33%

Atrial fibrillation is a common arrhythmia that may be persistent or paroxysmal. It is more common in women compared to men.

All patients with suspected atrial fibrillation should have a manual pulse check followed by a twelve-lead electrocardiogram. Those who have episodes less than 24 hours apart should have a 24-hour ambulatory electrocardiogram. In patients who experience episodes more than 24 hours apart, an event recorder electrocardiogram would be the most suitable investigation of choice.

24-hour blood pressure monitoring would be considered in a patient with suspected hypertension.

Electrocardiogram treadmill test is useful for revealing abnormal heart rhythms induced by exercise and ischaemic changes suggestive of coronary heart disease.

A 72-hour ambulatory electrocardiogram is used in much the same way as a 24 and 48-hour test. However, current NICE guidelines suggest for atrial fibrillation of episodes more than 24 hours apart an event recorder device is most useful.

Discuss (13) Improve

Next question

Atrial fibrillation: classification

An attempt was made in the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines to simplify and clarify the classification of atrial fibrillation (AF).

It is recommended that AF be classified into 3 patterns:

- first detected episode (irrespective of whether it is symptomatic or self-terminating)
- recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term **paroxysmal AF** is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term **persistent AF** is used. Such episodes usually last greater than 7 days
- in **permanent AF** there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 3

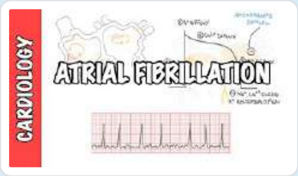
2021 Atrial fibrillation guidelines

Suggest link Report broken link

Media

Atrial fibrillation

Osmosis - YouTube 👍 2 👎 0


CARDIOLOGY
ATRIAL FIBRILLATION

Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 1 👎 1

Report broken media

Score: **18.9%**

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A 24-year-old man presents to the emergency department with one hour of palpitations and dizziness. He has no significant past medical history and is on no regular medications. He works as an accountant. There is no history of illicit drug use.

His observations are heart rate 180 beats per minute, blood pressure 88/59 mmHg, respiratory rate 22/minute, oxygen saturations 96% on air and temperature 36.4ºC.

Clinical examination is unremarkable except for a regular bounding tachycardia.

An ECG demonstrates ventricular tachycardia with positive concordance throughout the chest leads and similar morphology throughout.

As he is haemodynamically unstable, emergency electrical cardioversion is undertaken in the emergency department resuscitation area, which is successful.

Post cardioversion, his observations are heart rate 84 beats per minute, blood pressure 120/84 mmHg, respiratory rate 18/minute, oxygen saturations 96% on air and temperature 36.6ºC.

A repeat ECG demonstrates sinus rhythm and T wave inversion in leads V1-V3 with no other significant abnormalities.

Blood tests:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	188 * 10^9/L	(150 - 400)
WBC	7.2 * 10^9/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Urea	5.6 mmol/L	(2.0 - 7.0)

Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Adjusted calcium	2.41 mmol/L	(2.2 - 2.6)
Magnesium	0.92 mmol/L	(0.85 - 1.10)
Troponin	17 ng/L	(<14)

A CT pulmonary angiogram excludes a pulmonary embolism.

What is the likely diagnosis?

- ○ Arrhythmogenic right ventricular cardiomyopathy ×
- ○ Brugada syndrome ×
- ○ Catecholaminergic Polymorphic Ventricular Tachycardia ×
- ○ Right ventricular outflow tract ventricular tachycardia ×
- ○ Romano-Ward syndrome ×

Submit answer

Reference ranges ﹀

Score: **18.9%**

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Troponin	17 ng/L	(<14)

A CT pulmonary angiogram excludes a pulmonary embolism.

What is the likely diagnosis?

Arrhythmogenic right ventricular cardiomyopathy	64%
Brugada syndrome	23%
Catecholaminergic Polymorphic Ventricular Tachycardia	6%
Right ventricular outflow tract ventricular tachycardia	5%
Romano-Ward syndrome	2%

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Important for me Less important

Arrhythmogenic right ventricular cardiomyopathy (AVRC) is the correct answer. AVRC is a condition inherited with an autosomal dominant pattern, which is characterised by replacement of the normal right ventricular myocardium with fibrofatty tissue. It typically presents in the 2nd-4th decade of life with palpitations, syncope or sudden cardiac death and is a cause of monomorphic ventricular tachycardia. The resting ECG is typically abnormal and may demonstrate T wave inversion in V1-V3. The mildly elevated troponin in this case is simply secondary to ventricular tachycardia. Imaging will demonstrate structural and functional abnormalities of the right ventricle.

Brugada syndrome is incorrect. This is also an inherited cause of sudden cardiac death (autosomal dominant) but it traditionally causes a polymorphic ventricular tachycardia and the resting ECG is characterized by 'coved' ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave.

Catecholaminergic Polymorphic Ventricular Tachycardia is incorrect. This is an inherited cause of arrhythmia and sudden cardiac death characterized by palpitations, syncope of cardiac arrest precipitated by exercise or emotions. It is typically associated with polymorphic ventricular tachycardia as suggested by the name. Usually, the resting ECG is normal.

Right ventricular outflow tract ventricular tachycardia (RVOT-VT) is incorrect. Idiopathic RVOT-VT is not an inherited condition and the resting ECG is normal.

Romano-Ward syndrome is incorrect. This is a cause of congenital long QT syndrome, which can cause a polymorphic ventricular tachycardia. We are told there are no abnormalities on the resting ECG apart from T wave inversion. Therefore, we can assume the QT interval is normal.

👍 👎 Discuss (2) Improve

Next question >

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology
- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation

- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 13 👎 7

Arrhythmogenic right ventricular cardiomyopathy ECG

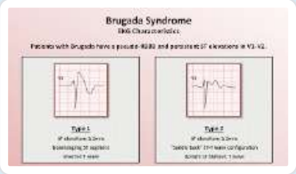
Suggest link Report broken link

Media

CARDIOLOGY
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome
EKG Characteristics

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

Report broken media

Score: **18.8%**

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Question 97 of 328

An 82-year-old man presents with loss of consciousness. His wife states that he complained of feeling dizzy, before going pale in colour and falling to the ground. He was unconscious for approximately one minute. On regaining consciousness he was not confused and recovered within minutes. He denies any chest pain, shortness of breath or palpitations. He states that over the past few months he has had several episodes of light headedness however he has never lost consciousness before.

A 7 day holter was performed:

Heart rate	3 episodes of bradycardia of 20-30 bpm which correlated with episodes of pre-syncope
Other events	Absence of P waves for 3.5 seconds

How will you manage this patient?

- ○ VVI pacemaker
- ○ Biventricular pacemaker
- ○ AAIR pacemaker
- ○ Reassurance
- ○ Bisoprolol

Submit answer

Reference ranges

Score: **18.8%**

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A 7 day holter was performed:

Heart rate	3 episodes of bradycardia of 20-30 bpm which correlated with episodes of pre-syncope
Other events	Absence of P waves for 3.5 seconds

How will you manage this patient?

VVI pacemaker	34%
Biventricular pacemaker	29%
AAIR pacemaker	35%
Reassurance	2%
Bisoprolol	0%

In pure sinus node dysfunction without AF or evidence of AV block, an AAIR or DDDR pacemaker can be used. Most cardiologists would choose a DDDR pacemaker since many of these patients go onto develop AV block

Important for me Less important

The 7 day holter confirms symptomatic sinus bradycardia and an episode of sinus arrest (absence of P wave for > 3 seconds). This confirms the diagnosis of sick sinus syndrome.

Sick sinus syndrome can be difficult to manage, as it can present with bradyarrhythmias, tachyarrhythmias, or a mixture of both. Bradyarrhythmias can be well controlled with pacemakers, while tachyarrhythmias respond well to rate limiting drugs such as calcium channel blockers, digoxin and beta blockers. However drugs to control tachyarrhythmia may exacerbate bradyarrhythmias. Therefore, a pacemaker is often implanted before drug therapy is begun for the tachyarrhythmia.

This patient has been having symptomatic bradycardia episodes secondary to sinoatrial node disease. Therefore an AAIR pacemaker is indicated. AAI pacemakers both sense and pace the atria. They are useful for isolated sinoatrial node disease. These are not suitable in patients with atrial fibrillation.

VVI pacemakers will both sense and pace the ventricle. VVI or VVR pacemakers are useful for pure sustained slow atrial fibrillation.

Biventricular pacemakers are used in heart failure with left bundle branch block.

Bisoprolol is a rate limiting drug which would worsen the bradycardia and the patients symptoms.

Discuss (10) | Improve

Next question >

Pacemaker: permanent ★

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:
- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):
- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)

- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):
- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):
- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:
- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:
- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that

chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

DUAL CHAMBER PACEMAKER
RIGHT ATRIUM AND
RIGHT VENTRICLE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **18.6%**

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A 45-year-old male patient presents with a worsening shortness of breath to the medical assessment unit. His has an accompanying letter from his GP stating that his past medical history is significant only for a deteriorating renal function that is secondary to polycystic kidney disease.

On examination there is no evidence of fluid overload, with the lung fields remaining clear and the JVP not elevated. You do however discern a late systolic murmur heard best at the cardiac apex which is immediately preceded by a 'click'.

Observations

- heart rate 82bpm
- blood pressure 106/86mmHg
- tespiratory rate 16 per minute
- temperature 36.5
- oxygen saturation 97% on room air

What is the most likely diagnosis?

○ Mitral valve prolapse

○ Mitral stenosis

○ Pulmonary stenosis

○ Mitral regurgitation

○ Aortic stenosis

Submit answer

Reference ranges

Question 98 of 328 ✓

A 45-year-old male patient presents with a worsening shortness of breath to the medical assessment unit. His has an accompanying letter from his GP stating that his past medical history is significant only for a deteriorating renal function that is secondary to polycystic kidney disease.

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Observations
- heart rate 82bpm
- blood pressure 106/86mmHg
- tespiratory rate 16 per minute
- temperature 36.5
- oxygen saturation 97% on room air

What is the most likely diagnosis?

Mitral valve prolapse	79%
Mitral stenosis	8%
Pulmonary stenosis	2%
Mitral regurgitation	8%
Aortic stenosis	4%

Adult polycystic kidney disease can be associated with extra-renal complications. Up to 85% of patients with autosomal dominant polycystic kidney disease (ADPKD) will exhibit liver cysts by the age of 30. Other complications include intra-cranial aneurysm and heart valve disease. Affected heart valves can occur in up to 25% of ADPKD patients and of them the majority have mitral valve prolapse. The majority of patients with mitral valve prolapse with be asymptomatic but when symptoms are present mitral valve prolapse has to be considered and investigated with an echocardiogram.

The 'click' coupled with the knowledge of the underlying ADPKD makes the murmur heard in this patient that of a mitral valve prolapse. The prolapse can worsen and cause a secondary mitral regurgitation leading to symptoms such as shortness of breath. The narrow pulse pressure present also suggests an element of left ventricular dysfunction.

👍 👎 💬 Discuss (4) Improve

Next question >

Mitral valve prolapse ★

Mitral valve prolapse is common, occurring in around 5-10 % of the population. It is usually idiopathic but may be associated with a wide variety of cardiovascular disease and other conditions

Associations
- congenital heart disease: PDA, ASD
- cardiomyopathy
- Turner's syndrome
- Marfan's syndrome, Fragile X
- osteogenesis imperfecta
- pseudoxanthoma elasticum
- Wolff-Parkinson White syndrome
- long-QT syndrome
- Ehlers-Danlos Syndrome
- polycystic kidney disease

Features
- patients may complain of atypical chest pain or palpitations
- mid-systolic click (occurs later if patient squatting)
- late systolic murmur (longer if patient standing)
- complications: mitral regurgitation, arrhythmias (including long QT), emboli, sudden death

123

Next question >

Textbooks

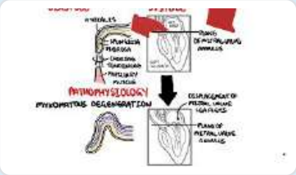
High-yield textbook

Extended textbook

Media

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 1 👎 0


PATHOPHYSIOLOGY
MYXOMATOUS DEGENERATION

Mitral valve prolapse

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **19.4%**

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Question 99 of 328

A 64-year-old caucasian male presented to heart failure clinic with shortness of breath on exertion. He has a history of heart failure, initially diagnosed three years previously.

His current medications included ramipril, aspirin, bisoprolol, simvastatin and spironolactone. He is concerned that despite all of these medications his breathlessness is worsening. In addition to this he is now using four pillows to sleep at night.

On examination he is comfortable at rest with fine crackles at both lung bases and pitting oedema reaching up to both knees. His observations reveal a respiratory rate of 16 breaths per minute, oxygen saturation 96% in room air, blood pressure 110/85 mmHg, heart rate 70 beats per minute, temperature 37.2ºC.

His ECG revealed a normal sinus rhythm with narrow QRS complexes. A recent echocardiogram completed two weeks previously demonstrated an ejection fraction of 30%. His current medications were reviewed and he was found to be on the maximum dose for each of these.

His baseline U&E results are as follows:

Na^+	136 mmol/L	(135 - 145)
K^+	4.5 mmol/L	(3.5 - 5.0)
Bicarbonate	26 mmol/L	(22 - 29)
Urea	6.7 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

What is the optimum treatment option for this patient?

○ Cardiac Resynchronisation Therapy

○ Digoxin

○ Hydralazine combined with a nitrate ×

○ Ivabradine ×

○ Sacubitril-Valsartan (after stopping ACE inhibitor) ×

Submit answer

Reference ranges ﹀

Score: **19.4%**

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Urea	6.7 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

What is the optimum treatment option for this patient?

Cardiac Resynchronisation Therapy	10%
Digoxin	2%

Hydralazine combined with a nitrate	3%
Ivabradine	9%
Sacubitril-Valsartan (after stopping ACE inhibitor)	76%

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & beta-blockers

Important for me Less important

This patient has heart failure, he is symptomatic despite maximum medical therapy with an ACE inhibitor, beta blocker and aldosterone antagonist. His heart rate is 70 bpm, therefore ivabradine cannot be used at this time. After a period of ACE inhibitor washout sacubitril-valsartan is the optimal therapeutic option.

Digoxin has been found to not have any improvement in long-term outcome and therefore is not the correct answer in this case.

Hydralazine with a nitrate is indicated in the afro-caribbean population; we are told this patient is caucasian, hence this answer is incorrect.

Ivabradine is contraindicated as the patients heart rate if 70 bpm, the guidelines state it should be > 75 bpm for ivabradine to be prescribed.

👍 👎 Discuss (5) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

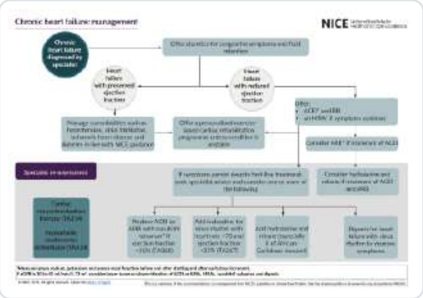
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan

    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction
Specialist treatments

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

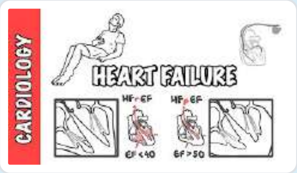
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

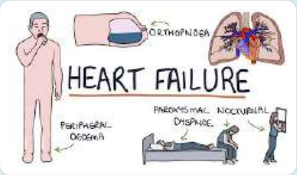
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HF↓EF
HF↑EF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube

👍 0 👎 2

Report broken media

Score: **20.2%**

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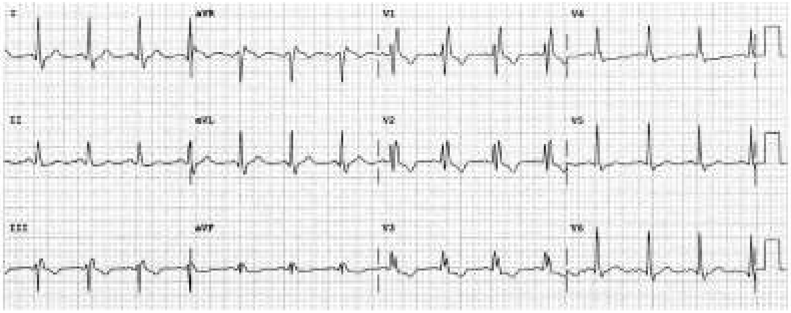
Question 100 of 328

A 50-year-old man is brought into the emergency department following a collapse whilst gardening. He denies any presyncopal symptoms but now describes shortness of breath. His past medical history includes prostate cancer and hypertension.

On examination, there is good air entry bilaterally. His peripheral pulses are present bilaterally with a capillary refill time of < 2 seconds. There is no postural drop in blood pressure and his neurological examination is unremarkable.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely cause of this patient's presentation?

○ Aortic stenosis

○ Hypercalcaemia

○ Hypokalaemia

○ Left ventricular hypertrophy

○ Pulmonary embolism

Submit answer

Reference ranges

Score: **20.2%**

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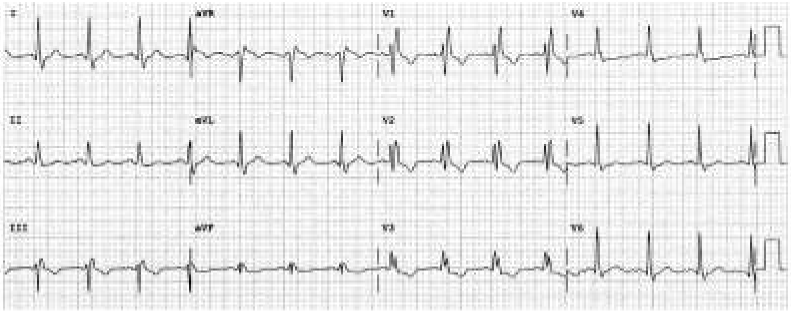


© Image used on license from Dr Smith, University of Minnesota

What is the most likely cause of this patient's presentation?

Aortic stenosis	12%
Hypercalcaemia	5%
Hypokalaemia	7%
Left ventricular hypertrophy	6%
Pulmonary embolism	71%

This patient's ECG demonstrates right bundle branch block (RBBB) with an RSR pattern in leads V1-V3 (creating an 'M-shaped' QRS complex), with wide slurred S waves in the leads I, aVL, V5-V6 (creating a 'W-shaped' QRS complex). A widened QRS interval (i.e. > 120ms) is also a feature of RBBB. There are multiple causes of RBBB and in some cases, it may be found incidentally as a normal variant. However, pathological causes commonly include increased right heart strain (e.g. right ventricular hypertrophy, cor pulmonale or pulmonary embolism). Given the history of malignancy, collapse and shortness of breath, a pulmonary embolism is the most likely cause of this patient's presentation to the hospital and ECG findings of RBBB.

Whilst aortic stenosis can cause the symptoms of collapse and breathlessness, it is not common for aortic stenosis to cause RBBB. Rather, aortic stenosis may be responsible for the appearance of left bundle branch block (LBBB) or left ventricular hypertrophy on an ECG.

The most common ECG finding in hypercalcaemia is QT interval shortening. In severe hypercalcaemia (> 3.4mmol/L) J waves may be seen. Although hypercalcaemia is more likely to be present in a patient with a diagnosis of malignancy, it would not be responsible for this patient's RBBB and is unlikely to be the underlying diagnosis.

Hypokalaemia causes the following ECG changes: prolonged PR interval, widespread ST depression and T wave flattening or inversion and prominent U waves (most clearly in leads V2-V3). It does not cause RBBB and is, therefore, incorrect.

Left ventricular hypertrophy (LVH) has numerous causes, most commonly hypertension. There are lots of specific voltage criteria to be met to accurately diagnose LVH on an ECG including:

- R wave in V4, V5 or V6 > 26mm
- R wave in V5 or V6 plus S wave in V1 > 35 mm
- Largest R wave plus largest S wave in precordial leads > 45 mm

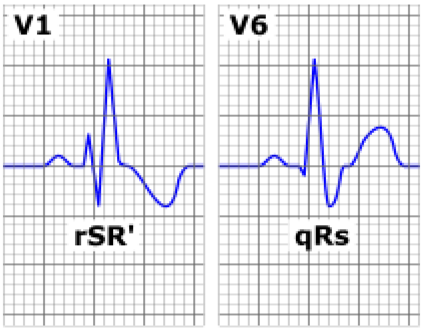
Additional features include left axis deviation and left atrial enlargement. Although this patient has hypertension, there are no features of LVH on his ECG and it is unlikely to cause his episode of syncope, making it the incorrect answer.

👍 👎 Discuss (3) Improve

Next question ›

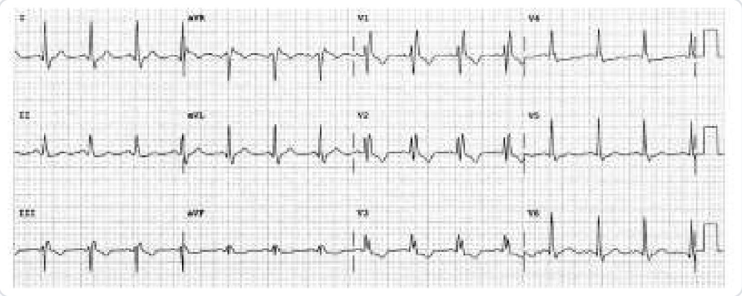
ECG: right bundle branch block

Right bundle branch block is a common feature seen on ECGs.



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6



Causes of RBBB

- normal variant - more common with increasing age
- right ventricular hypertrophy
- chronically increased right ventricular pressure - e.g. cor pulmonale
- pulmonary embolism
- myocardial infarction
- atrial septal defect (ostium secundum)
- cardiomyopathy or myocarditis

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

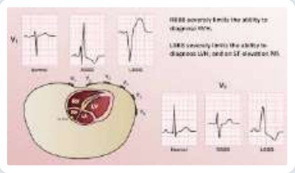
Life In The Fast Lane 👍 17 👎 5

Right Bundle Branch Block

Suggest link

Report broken link

Media



Intro to EKG Interpretation - Bundle Branch Blocks

Strong Medicine - YouTube 👍 2 👎 0

Bundle branch block

Osmosis - YouTube 👍 1 👎 0

Report broken media

Score: **21%**

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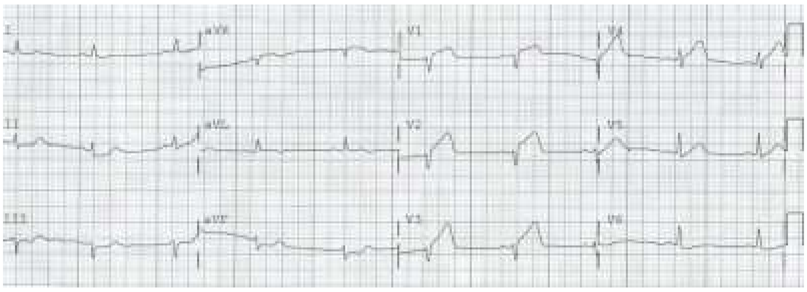
You review a 60-year-old man in resus who has been admitted with a 30 minute history of cardiac-sounding chest pain. His ECG is shown below:



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Where is the lesion most likely to be?

○ Right coronary

○ Left main stem

○ Posterior intraventricular artery

○ Left anterior descending

○ Left circumflex

Submit answer

Reference ranges

Score: **21%**

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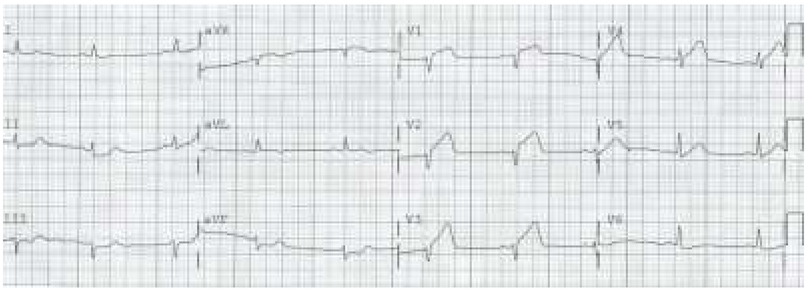
You review a 60-year-old man in resus who has been admitted with a 30 minute history of cardiac-sounding chest pain. His ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

Where is the lesion most likely to be?

Right coronary	5%
Left main stem	5%
Posterior intraventricular artery	1%
Left anterior descending	87%
Left circumflex	2%

Ischaemic changes in leads V1-V4 - left anterior descending

Important for me Less important

ST elevation can be seen in V2-V4 which is highly suggestive of a left anterior descending lesion. A left main stem occlusion would result in more widespread changes.

Discuss (3) Improve

Next question >

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

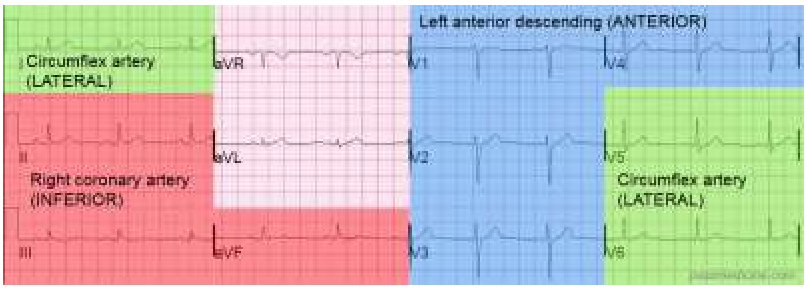

Circumflex artery
(LATERAL)
aVR
Left anterior descending (ANTERIOR)
V1
V4
II
aVL
V2
V5
Right coronary artery
(INFERIOR)
Circumflex artery
(LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

+ Q 123

Next question >

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 24 👎 40

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 14 👎 23

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **20.8%**

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A 53-year-old woman has been admitted to the hospital with progressively worsening dyspnoea. She denies a cough but describes orthopnoea and the feeling of palpitations.

Observations are shown below:

Heart rate	112 bpm
Blood pressure	86/42 mmHg
Respiratory rate	28 /min
Oxygen saturations	99%
Temperature	36.8 ºC

On examination, she is overweight and appears dyspnoeic. Her airway is self-maintained and her chest appears clear on auscultations. Heart sounds are difficult to auscultate, her JVP is raised and her capillary refill time is normal. There is no evidence of pedal oedema.

Given the likely diagnosis, what might you see on the ECG?

○ Deep lateral Q waves with left ventricular hypertrophy

○ Electrical alternans

○ Global ST elevation

○ S1 Q3 T3

○ Trifascicular block

Submit answer

Reference ranges

Score: **20.8%**

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Question 102 of 328 ✗

A 53-year-old woman has been admitted to the hospital with progressively worsening dyspnoea. She denies a cough but describes orthopnoea and the feeling of palpitations.

Observations are shown below:

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Respiratory rate	28 /min
Oxygen saturations	99%
Temperature	36.8 ºC

On examination, she is overweight and appears dyspnoeic. Her airway is self-maintained and her chest appears clear on auscultations. Heart sounds are difficult to auscultate, her JVP is raised and her capillary refill time is normal. There is no evidence of pedal oedema.

Given the likely diagnosis, what might you see on the ECG?

Deep lateral Q waves with left ventricular hypertrophy	7%
Electrical alternans	73%
Global ST elevation	4%
S1 Q3 T3	15%
Trifascicular block	2%

Electrical alternans is suggestive of cardiac tamponade

Important for me Less important

A history of dyspnoea, orthopnoea, and palpitations, associated with hypotension and tachycardia are classical symptoms of many cardiorespiratory illnesses. However, quiet or absent heart sounds with a raised JVP should raise concerns for cardiac tamponade. This can be confirmed with the ECG findings of sinus tachycardia, low QRS voltage, or electrical alternans.

Electrical alternans is a diagnostic feature of large pericardial effusion and cardiac tamponade. The alternate QRS amplitudes are due to the 'swinging' effect of the heart whilst contracting which subsequently gives rise to increased and decreased electrical impedance.

Deep lateral Q waves with left ventricular hypertrophy is a classical ECG finding of hypertrophic cardiomyopathy (HOCM). HOCM is the most common cause of sudden death in young people and is due to left ventricular hypertrophy without an identifiable cause. Patients can display all of the above symptoms. Dyspnoea is common due to left ventricular stiffness, resistance, and impaired filling. Patients may have a jerky pulse and may also present in arrhythmia, most commonly, atrial fibrillation. However, compared to the above scenario, patients have audible heart sounds, and may have a loud S1 and sometimes a 4th heart sound. There may be an ejection systolic murmur radiating to the apex which changes in intensity with a change in posture.

Pericarditis is associated with **global ST elevation**. Patients typically present with many of the above symptoms. Pericarditis is a common cause of pericardial effusion due to the accumulation of fluid in the pericardial sac due to inflammation. The distinguishing feature between pericarditis and pericardial effusion is the presence or absence of heart sounds, with pericarditis having audible heart sounds.

Symptoms of dyspnoea, palpitations, and orthopnoea, along with hypotension and tachycardia are common signs and symptoms associated with a pulmonary embolism (PE). The most common ECG finding in PE is sinus tachycardia, however, patients may have evidence of right axis deviation, atrial arrhythmia, right bundle branch block, P pulmonale, and **S1 Q3 T3**. S1 Q3 T3 is the presence of deep S waves in lead I along with Q waves and T wave inversion in lead 3.

Trifascicular block is the combination of third-degree atrioventricular block, right bundle branch block, and either left anterior fascicular block or left posterior fascicular block. These patients generally required pacemaker insertion. The most common causes are structural heart disease, particular fibrosis of the myocardium, and ischaemic heart disease. It may also occur in anterior myocardial infarction and aortic stenosis. Many of the pathological causes of trifascicular block may cause the symptoms described in the above scenario, however, heart sounds would still be audible. Trifascicular block is not associated with pericardial effusion.

👍 👎 Discuss (5) Improve

Next question >

Cardiac tamponade ★

Cardiac tamponade is characterized by the accumulation of pericardial fluid under pressure.

Classical features - Beck's triad:
- hypotension
- raised JVP
- muffled heart sounds

Other features:
- dyspnoea
- tachycardia
- an absent Y descent on the JVP - this is due to the limited right ventricular filling
- pulsus paradoxus - an abnormally large drop in BP during inspiration
- Kussmaul's sign - much debate about this
- ECG: electrical alternans

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

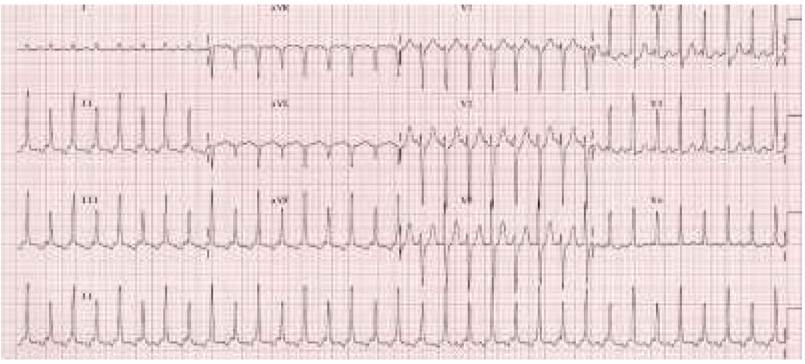
	Cardiac tamponade	**Constrictive pericarditis**
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present

	Cardiac tamponade	**Constrictive pericarditis**
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Management

- urgent pericardiocentesis



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An ECG demonstrating electrical alternans. Note the alternation of QRS complex amplitude between beats.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 12 👎 4

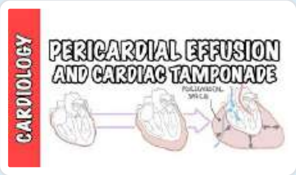
ECG Findings in Massive Pericardial Effusion

Suggest link Report broken link

Media

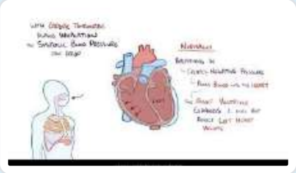
Echocardiogram of a Patient With Cardiac Tamponade

JAMA - YouTube 👍 12 👎 0


CARDIOLOGY
PERICARDIAL EFFUSION
AND CARDIAC TAMPONADE

Cardiac tamponade

Armando Hasudungan - YouTube 👍 1 👎 0



Cardiac tamponade

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **20.6%**

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Question 103 of 328

A 78-year-old woman attends the cardiology outpatient department after being referred by her general practitioner with angina. The patient reports consistent chest tightness after walking around 200 metres on flat ground but denies any episodes of pain at rest. Her symptoms have been stable at the current level for at least six months. Her GP has previously attempted treatment with both metoprolol and amlodipine but both were stopped after the patient reported dizziness and was found to be hypotensive.

The patient was formerly a heavy smoker but had stopped 15 years previously after suffering a non-ST elevation myocardial infarction (managed with medical therapy only). The patient had been diagnosed with type 2 diabetes mellitus 10 years previously and was known to have chronic kidney disease.

The patients current medications included aspirin 75 mg daily, simvastatin 20 mg daily, ramipril 1.25 mg daily and gliclazide 60 mg twice daily. She sometimes used a nitrate spray to relieve episodes of angina but found that it normally made her very dizzy.

Clinical examination was unremarkable except for a blood pressure of 98 / 65 mmHg.

Please see below for results of recent investigations.

Transthoracic echocardiogram: normal valvular function; moderately impaired left ventricular; ejection fraction 35 %.

Electrocardiogram: sinus rhythm at 85 beats per minute; normal axis; normal QRS complex, normal ST segments and T waves.

Urea	15.6 mmol / L
Creatinine	234 micromol / L
Sodium	135 mmol / L
Potassium	4.9 mmol / L

Cardiac stress MRI: evidence of mild myocardial ischaemia in left anterior descending territory, estimated < 10 % myocardium ischaemic.

What is the correct next line therapy for the patients angina?

- ○ Percutaneous coronary intervention ×
- ○ Nicorandil ×
- ○ Ranolazine ×
- ○ Ivabradine ×
- ○ Long-acting isosorbide mononitrate ×

Submit answer

Reference ranges ∨

Score: **20.6%**

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A 78-year-old woman attends the cardiology outpatient department after being referred by her general practitioner with angina. The patient reports consistent chest tightness after walking around 200 metres on flat ground but denies any episodes of pain at rest. Her symptoms have been stable at the current level for at least six months. Her GP has previously attempted treatment with both metoprolol and amlodipine but both were stopped after the patient reported dizziness and was found to be hypotensive.

The patient was formerly a heavy smoker but had stopped 15 years previously after suffering a non-ST elevation myocardial infarction (managed with medical therapy only). The patient had been diagnosed with type 2 diabetes mellitus 10 years previously and was known to have chronic kidney disease.

The patients current medications included aspirin 75 mg daily, simvastatin 20 mg daily, ramipril 1.25 mg daily and gliclazide 60 mg twice daily. She sometimes used a nitrate spray to relieve episodes of angina but found that it normally made her very dizzy.

Clinical examination was unremarkable except for a blood pressure of 98 / 65 mmHg.

Please see below for results of recent investigations.

Transthoracic echocardiogram: normal valvular function; moderately impaired left ventricular; ejection fraction 35 %.

Electrocardiogram: sinus rhythm at 85 beats per minute; normal axis; normal QRS complex, normal ST segments and T waves.

Urea	15.6 mmol / L
Creatinine	234 micromol / L
Sodium	135 mmol / L
Potassium	4.9 mmol / L

Cardiac stress MRI: evidence of mild myocardial ischaemia in left anterior descending territory, estimated < 10 % myocardium ischaemic.

What is the correct next line therapy for the patients angina?

Percutaneous coronary intervention	16%
Nicorandil	8%
Ranolazine	18%
Ivabradine	49%
Long-acting isosorbide mononitrate	10%

The patient has stable angina and has been intolerant of first line medical therapy. Due to her co-morbidities several of the second-line medical therapy options are contra-indicated. Ranolazine acts a late inward calcium channel antagonist but is contra-indicated in severe renal disease. Both nicorandil and ISMN are contra-indicated in this patient due to her history of hypotension.

Therefore, ivabradine is the most appropriate option. The drug acts as an If channel antagonist to reduce heart rate and is appropriate in this patient without a history of bradycardia. It should not be used in patients with moderate to severe angina as it has been shown to increase the incidence of cardiovascular events in these patients.

Revascularisation techniques such as PCI have not been show to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy options should be exhausted before consideration of invasive treatment (particularly in this patient with renal failure making contrast administration very undesirable).

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stunting in stable angina? BMJ 2016;352:i205.

👍 👎 Discuss (9) Improve

Next question >

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance

- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

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Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

Suggest link

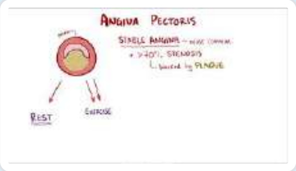
Report broken link

Media

MIDLINE STERNOTOMY SCAR
ANGINA
ATHEROSCLEROSIS
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

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Question 104 of 328

A 43 year old man has been referred to endocrinology clinic for blood pressure investigation and management. Over the past year the patient has had a persistently raised blood pressure between 170/100mmHg and 180/110mmHg. Despite starting the patient on ramipril 5mg once daily four weeks ago the patient's blood pressure on this visit remained raised at 164/93mmHg.

During the history taking part of the consultation the patient mentions that he has been troubled with headaches for the past year, and has noticed that his stool frequency has increased and his stools have become looser. He tells you that he's rather embarrassed to admit that he's been having flushing episodes, and feels that his clothes are much looser than they were 1 year ago.

On discussing his family history he mentions that both his mother and his father had to have cancerous lumps removed in their middle ages. His mother had a breast lump removed, and his father had some form of pancreatic mass removed.

On examination the patient is tall with a wide arm span. Examination of the cardiovascular system reveals only a minor tachycardia of 95bpm and a quiet systolic flow murmur not radiating to the carotids. Abdominal examination reveals no palpable masses, and examination of his lungs is completely normal.

The GP had previously arranged a 24h urinary catecholamine test; results are as follows:

Total urine catecholamines 210mcg/24hr. This is raised.

As a follow up to this test the GP had arranged a CT of the patient's abdomen and pelvis which is reported as normal apart from a few incidental simple renal cysts.

Urinalysis in clinic today shows:

Leucocytes	-
Blood	-

Glucose	+
Ketones	-

Which test is most likely to elucidate the cause of the patient's hypertension?

- ○ Pentagastrin stimulation test
- ○ 24h urinary HIAA
- ○ MRI renal angiography
- ○ GH, IGF-1 and 'gut hormones'
- ○ MIBG (metaiodobenzylguanidine) scan

Submit answer

Reference ranges

Score: **20.4%**

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Urinalysis in clinic today shows:

Leucocytes	-
Blood	-

Glucose	+
Ketones	-

Which test is most likely to elucidate the cause of the patient's hypertension?

Pentagastrin stimulation test	5%
24h urinary HIAA	49%
MRI renal angiography	8%
GH, IGF-1 and 'gut hormones'	9%
MIBG (metaiodobenzylguanidine) scan	29%

This question tests the candidates knowledge of causes of secondary hypertension. The commonly listed causes of secondary hypertensions are listed below:

Endocrine	**Adrenal**	**Renal**
Cushing's Syndrome	Conn's Syndrome	Diabetic nephropathy
Acromegaly	Adrenal hyperplasia	Chronic glomerulonephritis/tubul nephritis
Thyroid disease	Phaeochromocytoma	Adult polycystic kidney d
Hyperparathyroidism	-	Renovascular disease
-	-	-

The best way of narrowing down to the correct answer in this question is to actually look at what the answers are suggesting:

Pentagastrin stimulation test	Test for medullary carcinoma of the thyroid (associated with MEN2 - multiple endocrine neoplasia)
24h Urinary HIAA	Screening for carcinoid
MRI renal angiography	Test for renal artery stenosis
GH, IGF-1 and 'gut hormones'	Test for pancreatic malignancy associated with MEN1
MIBG scan	Test for phaeochromocytoma

While this patient might well have MEN2 (as suggested by the patients body habitus and features consistent with phaeochromocytoma), medullary thyroid cancer would be unlikely to cause his blood pressure issues. Therefore the pentagastrin stimulation test, where one injects the patient with pentagastrin and looks for an abnormally high release of calcitonin as a response, is not a suitable first test. It may well be considered if the patient is diagnosed with a phaeochromocytoma to rule out medullary thyroid cancer as part of MEN 2.

The patient does have a few features consistent with carcinoid syndrome, for example the episodic flushing and increased stool frequency. However carcinoid syndrome doesn't present with these symptoms until it has metastasised to the liver, and this patient has had a normal CT scan of his abdomen making this a less likely diagnosis.

MRI renal angiography is used to diagnose renal artery stenosis; this patient has far too many other endocrine features for this to be renal artery stenosis. In addition the fact his blood pressure has not got worse with angiotensin converting enzyme inhibitors makes the diagnosis less likely.

Testing for gut hormones would be done in a patient in whom you were considering a diagnosis of pancreatic neoplasm as part of MEN1.

This leaves the MIBG (metaiodobenzylguanidine) scan. The patient has raised levels of total urinary catecholamines, suggesting a diagnosis of phaeochromocytoma, however, his CT scan show's no evidence. As you may be aware there is a rule of 10 that is commonly used to describe phaeochromocytoma, in that:
10% are malignant
10% are bilateral
10% are familial
10% are in children

10% are extra-adrenal

The other sites that phaeochromocytomas can present include: Anywhere alone the sympathetic chain which runs alongside the spinal cord, the distal aorta, ureters and urinary bladder.

The MIBG scan uses radioactive iodine as a tracer for phaeochromocytoma tumour cells, which is detected through the use of a gamma camera., making it the next test that should be used in diagnosing this patient's phaeochromocytoma, i.e. the cause of his hypertension.

Kumar & Clark's Clinical Medicine Eight Edition page 778

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Next question >

Hypertension: secondary causes

It is thought that between 5-10% of patients diagnosed with hypertension have **primary hyperaldosteronism**, including Conn's syndrome. This makes it the single most common cause of secondary hypertension.

Renal disease accounts for a large percentage of the other cases of secondary hypertension. Conditions which may increase the blood pressure include:

- glomerulonephritis
- pyelonephritis
- adult polycystic kidney disease
- renal artery stenosis

Endocrine disorders (other than primary hyperaldosteronism) may also result in increased blood pressure:

- phaeochromocytoma
- Cushing's syndrome
- Liddle's syndrome
- congenital adrenal hyperplasia (11-beta hydroxylase deficiency)
- acromegaly

Drug causes:

- steroids
- monoamine oxidase inhibitors
- the combined oral contraceptive pill
- NSAIDs
- leflunomide

Other causes include:

- pregnancy
- coarctation of the aorta

+ Q 123

Next question >

Textbooks

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Media

Hypertension

Osmosis - YouTube 3 0

Report broken media

Score: **20.2%**

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104 ✕

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An 84-year-old male was admitted to the coronary care unit (CCU) after being admitted as a primary percutaneous coronary intervention (PPCI) call for sudden onset chest pain and ST elevation on anterior leads of his ECG. Angiography demonstrated a mid-left anterior descending artery acute occlusion likely secondary to plaque rupture that was stented with a drug-eluting stent. Two hours after being admitted to CCU, he complained of nausea and vomited twice, which settled after being prescribed cyclizine by a passing medical senior house officer. Thirty minutes afterwards, you are asked to see the patient as the patient has become increasingly short of breath. He complains of no new chest pain, nausea, vomiting, sweating or palpitations. Interrogation of his cardiac telemetry reveals a new sinus tachycardia only without other arrhythmias. On examination, the patient is in respiratory distress, with bibasal inspiratory crackles and raised jugular venous pulse.

His observations are as follows: blood pressure is 186/80 mmHg, heart rate 120 beats per minute and regular, Sats 92% on 3 litres, respiratory rate 33/min.

A repeat 12-lead ECG reveals no new ST elevation changes or other ischaemic changes. A portable chest radiograph demonstrates bibasal alveolar oedema and bilateral small pleural effusions. What is the cause of the patient's deterioration?

- ○ In-stent thrombosis
- ○ Pulmonary embolus
- ○ New independent cardiac ischaemic occlusion
- ○ Cyclizine induced heart failure
- ○ Cardiac tamponade secondary to cardiac instrumentation

Submit answer

An 84-year-old male was admitted to the coronary care unit (CCU) after being admitted as a primary percutaneous coronary intervention (PPCI) call for sudden onset chest pain and ST elevation on anterior leads of his ECG. Angiography demonstrated a mid-left anterior descending artery acute occlusion likely secondary to plaque rupture that was stented with a drug-eluting stent. Two hours after being admitted to CCU, he complained of nausea and vomited twice, which settled after being prescribed cyclizine by a passing medical senior house officer. Thirty minutes afterwards, you are asked to see the patient as the patient has become increasingly short of breath. He complains of no new chest pain, nausea, vomiting, sweating or palpitations. Interrogation of his cardiac telemetry reveals a new sinus tachycardia only without other arrhythmias. On examination, the patient is in respiratory distress, with bibasal inspiratory crackles and raised jugular venous pulse.

His observations are as follows: blood pressure is 186/80 mmHg, heart rate 120 beats per minute and regular, Sats 92% on 3 litres, respiratory rate 33/min.

A repeat 12-lead ECG reveals no new ST elevation changes or other ischaemic changes. A portable chest radiograph demonstrates bibasal alveolar oedema and bilateral small pleural effusions. What is the cause of the patient's deterioration?

In-stent thrombosis	10%
Pulmonary embolus	13%
New independent cardiac ischaemic occlusion	2%
Cyclizine induced heart failure	54%
Cardiac tamponade secondary to cardiac instrumentation	22%

This patient has entered acute pulmonary oedema, associated with systemic hypertension and tachycardia following administration of cyclizine in the context of an acute STEMI, typical of cyclizine-induced heart failure. Cyclizine is a H1 receptor antagonist and anticholinergic, resulting in systemic hypertension and inducing tachycardia. It can aggravate a fragile myocardium into acute failure and is hence not recommended by NICE in patients with ACS or severe heart failure1. In this patient, the lack of chest pain or ST elevation on ECG makes acute in-stent thrombosis or a further ACS episode unlikely. The patient is hypertensive instead of hypotensive, with no mention of low voltage on ECG, making cardiac tamponade an unlikely cause.

1. NICE evidence 2013 'Can cyclizine be given to a patient who has an MI?'

👍 | 👎 | Discuss (16) | Improve

Next question >

Myocardial infarction: STEMI management

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given

- **aspirin**
- **P2Y12-receptor antagonist**. Clopidogrel was the first P2Y12-receptor antagonist to be widely used but now ticagrelor is often favoured as studies have shown improved outcomes compared to clopidogrel, but at the expense of slightly higher rates of bleeding. This approached is supported in SIGN's 2016 guidelines. They also recommend that prasugrel (another P2Y12-receptor antagonist) could be considered if the patient is going to have a percutaneous coronary intervention
- **unfractionated heparin** is usually given for patients who're are going to have a PCI. Alternatives include low-molecular weight heparin

NICE suggest the following in terms of oxygen therapy:

- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:

- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is available.

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres. Thrombolysis should be performed in patients without access to primary PCI

With regards to thrombolysis:

- tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase
- tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation

- if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis
- for patients successfully treated with thrombolysis PCI has been shown to be beneficial. The optimal timing of this is still under investigation

Glycaemic control in patients with diabetes mellitus

- in 2011 NICE issued guidance on the management of hyperglycaemia in acute coronary syndromes
- it recommends using a dose-adjusted insulin infusion with regular monitoring of blood glucose levels to glucose below 11.0 mmol/l
- intensive insulin therapy (an intravenous infusion of insulin and glucose with or without potassium, sometimes referred to as 'DIGAMI') regimes are not recommended routinely

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Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 6 👎 9

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

SIGN 👍 7 👎 8

2016 Acute coronary syndrome guidelines

NICE 👍 10 👎 7

2011 Managing hyperglycaemia in acute coronary syndromes

Suggest link Report broken link

Score: **20%**

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105	✗

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A 63-year-old patient is called to his GP for a health check. He has a past medical history of asthma and a family history of hypertension and bowel cancer. He has a 10 pack-year history of smoking but quit with the help of the practice nurse 2 years ago. He drinks 24 units of alcohol per week, and his body mass index is 25 kg/m². He does not take any regular medication. His blood pressure is 155/95 mmHg. He is sent for ambulatory blood pressure monitoring (ABPM), which shows an average blood pressure of 145/90 mmHg. His 10-year cardiovascular risk is 8%.

Examination, including fundoscopy, is unremarkable.

Bloods show:

Hb	132 g/l
Platelets	330 * 10^9/l
WBC	7.3 * 10^9/l

Na^+	142 mmol/l
K^+	3.9 mmol/l
Urea	5 mmol/l
Creatinine	80 µmol/l
eGFR	91 ml/min
Fasting plasma glucose	5mmol/L
Serum total cholesterol	4mmol/L
HDL cholesterol	1.8mmol/L

Urinary albumin:creatinine ratio	1.1 mg/mmol
Urine dip	negative for haematuria

An ECG shows sinus rhythm.

What is the first step in the management of this man's blood pressure?

- ○ Lisinopril ×
- ○ Lifestyle advice ×
- ○ Amlodipine ×
- ○ Spironolactone ×
- ○ Atenolol ×

Submit answer

Reference ranges ⌄

Score: **20%**

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Question 106 of 328 ✕

A 63-year-old patient is called to his GP for a health check. He has a past medical history of asthma and a family history of hypertension and bowel cancer. He has a 10 pack-year history of smoking but quit with the help of the practice nurse 2 years ago. He drinks 24 units of alcohol per week, and his body mass index is 25 kg/m². He does not take any regular medication. His blood pressure is 155/95 mmHg. He is sent for ambulatory blood pressure monitoring (ABPM), which shows an average blood pressure of 145/90 mmHg. His 10-year cardiovascular risk is 8%.

Examination, including fundoscopy, is unremarkable.

Bloods show:

Hb	132 g/l
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Creatinine	80 µmol/l
eGFR	91 ml/min
Fasting plasma glucose	5mmol/L
Serum total cholesterol	4mmol/L
HDL cholesterol	1.8mmol/L

Urinary albumin:creatinine ratio	1.1 mg/mmol
Urine dip	negative for haematuria

An ECG shows sinus rhythm.

What is the first step in the management of this man's blood pressure?

Lisinopril	20%
Lifestyle advice	38%
Amlodipine	41%
Spironolactone	1%
Atenolol	1%

This patient has a persistently raised blood pressure. During ABPM, two measurements are taken every hour during waking hours and at least 14 measurements are used to confirm the average blood pressure. This man's average blood pressure is >135/85 mmHg on ABPM; he has stage 1 hypertension, which warrants intervention. Given that his 10-year cardiovascular risk is low, and there is no evidence of end-organ damage, the most appropriate option at this stage is lifestyle advice.

The patient should be advised regarding diet and exercise. He has already given up smoking, but he should cut down on caffeine, salt and alcohol. Relaxation therapies have been shown to produce a small reduction in blood pressure, but these are not routinely offered in primary care. Drug treatment should be initiated if the average blood pressure is >150/95 mmHg, if he has a 10-year cardiovascular risk >10%, or if he develops end-organ damage. The first drug of choice in a patient over 55 years would be a calcium-channel blocker, such as amlodipine.

Discuss (6) | Improve

Next question >

Hypertension: management

NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

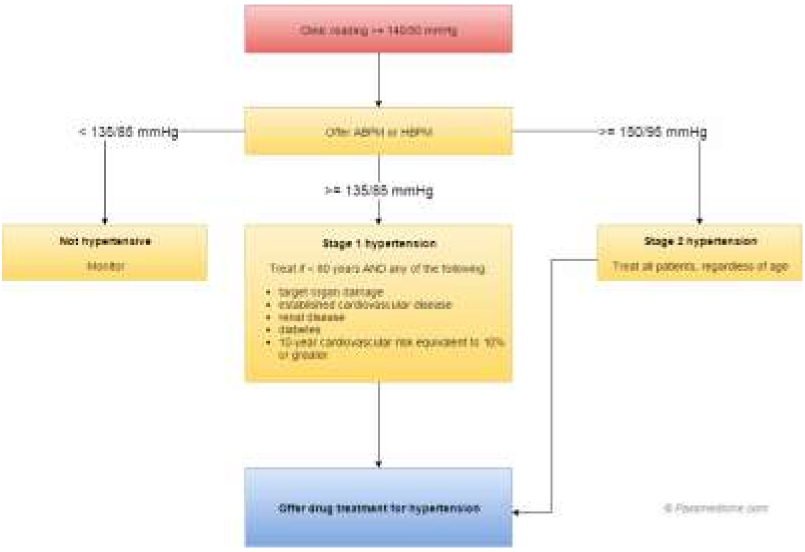
- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated

- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	**Criteria**
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
• target organ damage
• established cardiovascular disease
• renal disease
• diabetes
• 10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

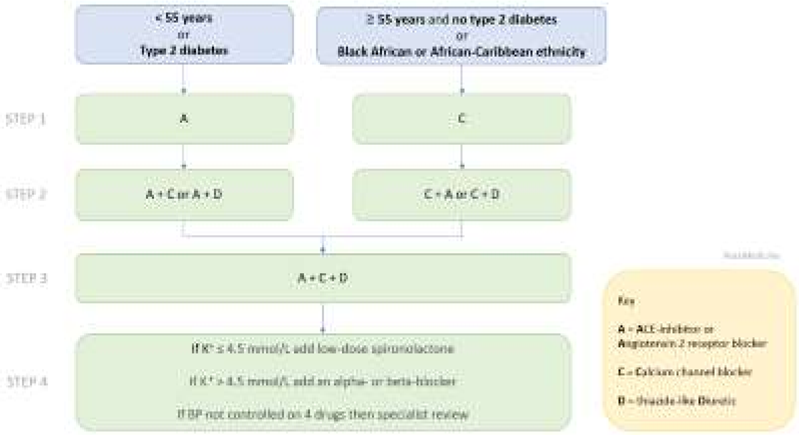
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
  - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

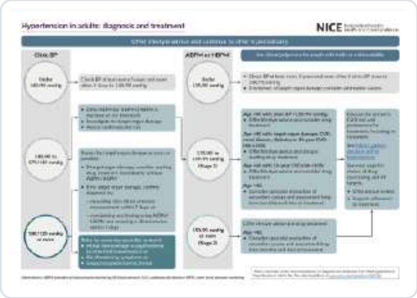
- add a third drug to make, i.e.:
  - if already taking an **(A + C)** then add a **D**
  - if already **(A + D)** then add a **C**
- **(A + C + D)**

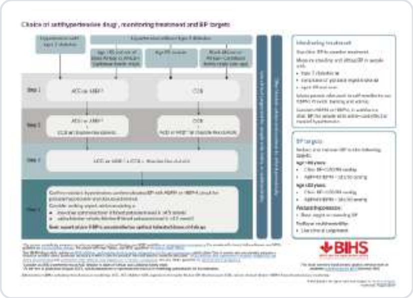
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
  - confirm elevated clinic BP with ABPM or HBPM
  - assess for postural hypotension.
  - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*





Blood pressure targets

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

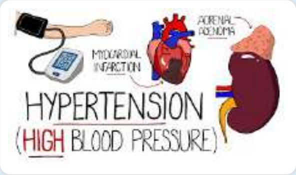
👍 162 👎 162

2019 Hypertension guidelines

Suggest link

Report broken link

Media


MYOCARDIAL INFARCTION
ADRENAL ADENOMA
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **19.8%**

1 ✗
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Question 107 of 328

A 23-year-old rugby player has a blackout whilst playing a match. No head injury is noted. There was no warning and he recovered quickly within seconds. This has never happened before. There was no chest pain. His father died in his sleep aged 49 and was known to have Brugada syndrome. At presentation several hours later the patient's electrocardiogram (ECG) and routine blood profile including FBC, U&E, and glucose are normal. He is sent to cardiology clinic for further investigation. A diagnosis of Brugada syndrome is suspected. Which agent may they elect to use there to provoke the down-going ST elevation in leads V1-V3 characteristically seen in the ECG of a patient with Brugada Syndrome?

○ Digoxin ×

○ Sotalol ×

○ Nifedipine ×

○ Adenosine ×

○ Flecanide ×

Submit answer

Reference ranges

Score: **19.8%**

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A 23-year-old rugby player has a blackout whilst playing a match. No head injury is noted. There was no warning and he recovered quickly within seconds. This has never happened before. There was no chest pain. His father died in his sleep aged 49 and was known to have Brugada syndrome. At presentation several hours later the patient's electrocardiogram (ECG) and routine blood profile including FBC, U&E, and glucose are normal. He is sent to cardiology clinic for further investigation. A diagnosis of Brugada syndrome is suspected. Which agent may they elect to use there to provoke the down-going ST elevation in leads V1-V3 characteristically seen in the ECG of a patient with Brugada Syndrome?

Digoxin	5%
Sotalol	20%
Nifedipine	4%
Adenosine	11%
Flecanide	61%

Exertional syncope in a young person with a family history of sudden cardiac death or with an abnormal ECG at presentation should trigger alarm bells in your head this person may be at risk of sudden cardiac death (which you should note is a preventable death if the condition is recognised in time and an implantable cardioverter defibrillator (ICD) is put in.)

The condition alluded to here is Brugada Syndrome. This is an autosomal dominant condition resulting in loss of function of the cardiac sodium channel. The characteristic ECG change is down-going ST elevation in V1 to V3. However, this may not be present, especially so long after the syncope episode. The way to bring out the ECG change in cardiology clinic safely is to give a sodium channel blocker (usually flecainide), providing additional sodium channel blockade to that already pathologically present. Once a diagnosis is made, the life-saving treatment is an ICD device. Genetic family counselling and testing will be of additional importance (testing for a mutation in the SCN5A gene).

The substantial majority of the approximately 100,000 sudden cardiac deaths each year in the UK are caused by coronary heart disease. However, most sudden deaths in people under 30 years old are caused by inherited cardiomyopathies and arrhythmias. A sensible list of differentials to consider in a young-person sudden cardiac death case are hypertrophic obstructive cardiomyopathy (HOCM), long-QT syndrome, Brugada, and arrhythmogenic right ventricular cardiomyopathy (ARVC). These will each be covered elsewhere using different questions as triggers.

ECG changes and further reading available at:
http:www.csanz.edu.au/documents/guidelines/clinicalpractice/BrugadaSyndrome.pdf

👍 👎 Discuss (1) Improve

Next question >

Brugada syndrome

Brugada syndrome is a form of inherited cardiovascular disease with may present with sudden cardiac death. It is inherited in an autosomal dominant fashion and has an estimated prevalence of 1:5,000-10,000. Brugada syndrome is more common in Asians.

Pathophysiology

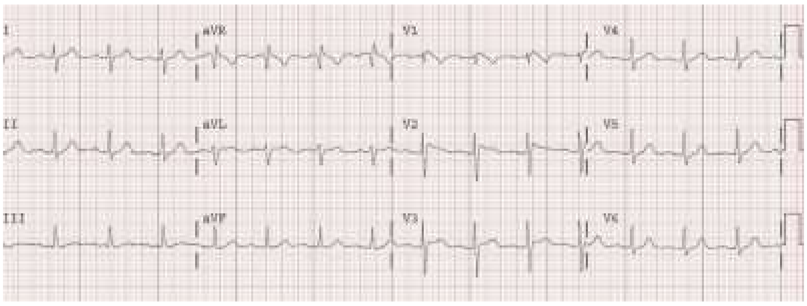
- a large number of variants exist
- around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the myocardial sodium ion channel protein

ECG changes

- convex ST segment elevation > 2mm in > 1 of V1-V3 followed by a negative T wave
- partial right bundle branch block
- the ECG changes may be more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases of Brugada syndrome

Management

- implantable cardioverter-defibrillator



© Image used on license from Dr Smith, University of Minnesota

ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

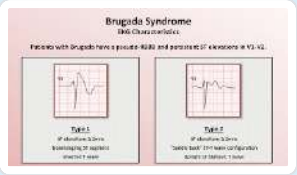
Life in the fast lane	👍 29	👎 5

Brugada syndrome

Suggest link

Report broken link

Media


Brugada Syndrome

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 12 👎 0

SODIUM CHANNEL (SCN5A)
BRUGADA SYNDROME
ECG PLACEMENT
BRUGADA PATTERN
RIGHT VENTRICLE ORIGIN

Brugada syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **19.6%**

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106	✗
107	✗

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Question 108 of 328

A 65-year-old lady with a history of diabetes and newly diagnosed atrial fibrillation (AF) was admitted to hospital with a moderate-large sized embolic stroke. Her blood pressure on admission was 165/90 mmHg with a heart rate of 95 beats per minute.

An MRI brain was performed 24 hours after admission which showed a moderate to large area of infarction involving the anterior 2/3 of the left middle cerebral artery territory without haemorrhagic transformation.

With regards to management of her AF and stroke prevention, the most appropriate decision would be to commence which of the following?

- ○ An adjusted dose intravenous heparin infusion ×
- ○ Therapeutic dose low molecular weight heparin (LMWH) and transition to oral anticoagulation in 10-14 days ×
- ○ Aspirin and wait 10-14 days before commencing formal anticoagulation ×
- ○ Warfarin with LMWH bridging therapy ×
- ○ OR calculate the CHADS-vasc score and make a decision ×

Submit answer

Reference ranges

Score: **19.6%**

1 ×
2 ×

3	✗
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Question 108 of 328 ✗

A 65-year-old lady with a history of diabetes and newly diagnosed atrial fibrillation (AF) was admitted to hospital with a moderate-large sized embolic stroke. Her blood pressure on admission was 165/90 mmHg with a heart rate of 95 beats per minute.

An MRI brain was performed 24 hours after admission which showed a moderate to large area of infarction involving the anterior 2/3 of the left middle cerebral artery territory without haemorrhagic transformation.

With regards to management of her AF and stroke prevention, the most appropriate decision would be to commence which of the following?

An adjusted dose intravenous heparin infusion	1%
Therapeutic dose low molecular weight heparin (LMWH) and transition to oral anticoagulation in 10-14 days	13%
Aspirin and wait 10-14 days before commencing formal anticoagulation	76%
Warfarin with LMWH bridging therapy	3%
OR calculate the CHADS-vasc score and make a decision	7%

Moderate-large areas of cerebral infarction are subject to the risk of haemorrhagic transformation within the acute period - usually defined as 10-14 days. With this in mind, patients with AF should be cautiously commenced on therapeutic dose formal anticoagulation 10-14 days after stroke onset in the absence of spontaneous haemorrhagic transformation.

The use of aspirin and/or low dose heparin/LMWH for venous thromboembolism (VTE) prophylaxis can be considered within 24 hours of symptom onset.

CHADS2 or CHADS-Vasc scores are used to estimate the risk of embolic stroke in patients with atrial fibrillation and assist with deciding on whether or not to anti-coagulate a patient. This patient scores highly enough to be started on formal anticoagulation, but this is not the focus of the question in this case. The focus is on the timing of anticoagulation in this clinical setting.

👍 👎 Discuss (5) Improve

Next question >

Atrial fibrillation: anticoagulation

NICE updated their guidelines on the management of atrial fibrillation (AF) in 2021.

Does the patient need anticoagulation?

The need for anticoagulation should be considered in patients with any history of AF, not just if they are in AF currently, i.e. the following groups should be assessed:

- symptomatic or asymptomatic paroxysmal, persistent or permanent atrial fibrillation
- atrial flutter
- a continuing risk of arrhythmia recurrence after cardioversion back to sinus rhythm or catheter ablation

NICE suggest using the **CHA_2DS_2-VASc** score to determine the most appropriate anticoagulation strategy. This scoring system superceded the $CHADS_2$ score.

	Risk factor	**Points**
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A_2	Age >= 75 years	2
	Age 65-74 years	1
D	Diabetes	1

	Risk factor	Points
S_2	Prior Stroke, TIA or thromboembolism	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
S	Sex (female)	1

The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender)
2 or more	Offer anticoagulation

Remember that if a CHA_2DS_2-VASc score suggests no need for anticoagulation it is important to ensure a transthoracic echocardiogram has been done to exclude valvular heart disease, which in combination with AF is an absolute indication for anticoagulation.

Assessing bleeding risk

Doctors have always thought carefully about the risk/benefit profile of starting someone on warfarin. A history of falls, old age, alcohol excess and a history of previous bleeding are common things that make us consider whether anticoagulation is in the best interests of the patient., but NICE warn us not to withhold anticoagulation solely on the grounds of age or risk falls. NICE now recommend we formalise this risk assessment using the ORBIT scoring system. Previously the HAS-BLED scoring system was recommended.

Variable	Points
Haemoglobin <130 g/L for males and < 120 g/L for females, or haemtocrit < 40% for males and < 36% for females	2
Age > 74 years	1
Bleeding history (GI bleeding, intracranial bleeding or haemorrhagic stroke)	2
Renal impairment (GFR < 60 mL/min/1.73m^2)	1
Treatment with antiplatelet agents	1

There are no formal rules on how we act on the ORBIT score and individual patient factors should be taken into account. However, the following table acts as a guide:

ORBIT score	Risk group	Bleeds per 100 patient-years
0-2	Low	2.4
3	Medium	4.7
4-7	High	8.1

If treatment is indicated, which anticoagulant?

For many years warfarin was the anticoagulant of choice in atrial fibrillation. This changed following the development of **direct oral anticoagulants (DOACs)**. These have the advantage of not requiring regular blood tests to check the INR and now recommended as the first-line anticoagulant for patients with AF. If a patient was previously started on warfarin, NICE recommend discussing switching to a DOAC during a routine follow-up appointment.

The following DOACs are recommended by NICE for reducing stroke risk in AF:
- apixaban
- dabigatran
- edoxaban

- rivaroxaban

Warfarin is now used second-line, in patients where a DOAC is contraindicated or not tolerated.

Aspirin is not recommended for reducing stroke risk in patients with AF.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 13 👎 14

2020 Atrial fibrillation guidelines

NICE 👍 18 👎 17

2019 Stroke and transient ischaemic attack in over 16s: diagnosis and initial management

SPARCtool 👍 28 👎 8

Stroke Prevention in Atrial Fibrillation Risk Tool

NICE

👍 43 👎 9

2021 Atrial fibrillation guidelines

Suggest link

Report broken link

Score: **19.4%**

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Question 109 of 328

A 57-year-old man attends his GP surgery for review. For the past year, he has experienced exertional chest pain, which initially only came on whilst walking uphill, but has now started to affect him when walking on the flat. The pain is alleviated by the use of glyceryl trinitrate spray. He denies ever having had chest pain at rest and does not report any symptoms of postural hypotension. His other medications include aspirin, atorvastatin and atenolol, which has been maximally titrated.

What is the most appropriate next step in management for this patient?

- ○ Refer for assessment for percutaneous coronary intervention (PCI) or CABG ×
- ○ Start amlodipine ×
- ○ Start isosorbide mononitrate ×
- ○ Start ranolazine ×
- ○ Start verapamil ×

Submit answer

Reference ranges ∨

Score: **19.4%**

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A 57-year-old man attends his GP surgery for review. For the past year, he has experienced exertional chest pain, which initially only came on whilst walking uphill, but has now started to affect him when walking on the flat. The pain is alleviated by the use of glyceryl trinitrate spray. He denies ever having had chest pain at rest and does not report any symptoms of postural hypotension. His other medications include aspirin, atorvastatin and atenolol, which has been maximally titrated.

What is the most appropriate next step in management for this patient?

Refer for assessment for percutaneous coronary intervention (PCI) or CABG	13%
Start amlodipine	63%
Start isosorbide mononitrate	13%
Start ranolazine	5%
Start verapamil	6%

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

Important for me Less important

The correct answer is **start amlodipine**. This patient has a likely diagnosis of stable angina, as he suffers from exertional chest pain which is alleviated by the use of GTN spray. As he is currently taking a beta-blocker at the maximum permissible dose, the next most appropriate step in management is to add in a calcium channel blocker. In this scenario, dihydropyridine calcium channel blockers are preferred to non-dihydropyridine calcium channel blockers, due to the increased risk of the latter causing complete heart block in combination with beta-blockers.

Refer for assessment for percutaneous coronary intervention (PCI)

or CABG is incorrect. As the patient has stable angina, with no symptoms at rest, he does not warrant urgent referral for PCI. Non-urgent referral for PCI or CABG would be appropriate if his symptoms were not improved by the addition of amlodipine.

Start isosorbide mononitrate is incorrect. This would be an appropriate third-line management option for stable angina in this patient if amlodipine failed to alleviate his symptoms. However, it should only be started whilst awaiting assessment for revascularization with PCI or CABG.

Start ranolazine is incorrect. This would also be more appropriate as a third-line management option whilst awaiting specialist assessment for PCI or CABG.

Start verapamil is incorrect. Although it would be reasonable to start a calcium channel blocker in this patient, starting a non-dihydropyridine calcium channel blocker such as verapamil in a patient already on a beta-blocker would run the risk of precipitating complete heart block.

👍 | 👎 | 💬 Discuss (2) | Improve

Next question ›

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-

release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

Suggest link

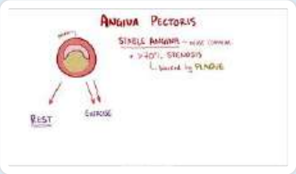
Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **19.3%**

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A 63-year-old man presents for review at his GP surgery. He is known to have hypertension and was started on amlodipine five years ago. Due to persistent poor hypertensive control, he began taking enalapril two years ago but has continued to struggle to keep his blood pressure within the required range. He reports no symptoms of postural hypotension, and his latest ambulatory blood pressure readings show an average blood pressure of 161/93 mmHg.

His latest blood test results are shown below:

Na^+	138 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)

What medication should be offered to this patient as the next step in managing his hypertension?

○ Bendroflumethiazide

○ Bisoprolol

○ Doxazosin

○ Indapamide

○ Spironolactone

Submit answer

Reference ranges

Score: **19.3%**

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A 63-year-old man presents for review at his GP surgery. He is known to have hypertension and was started on amlodipine five years ago. Due to persistent poor hypertensive control, he began taking enalapril two years ago but has continued to struggle to keep his blood pressure within the required range. He reports no symptoms of postural hypotension, and his latest ambulatory blood pressure readings show an average blood pressure of 161/93 mmHg.

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Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)

What medication should be offered to this patient as the next step in managing his hypertension?

Bendroflumethiazide	27%
Bisoprolol	3%
Doxazosin	2%
Indapamide	60%
Spironolactone	8%

Poorly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

Important for me Less important

The correct answer is **indapamide**. Current guidelines advise that in the absence of any clear contraindications, patients with poorly controlled hypertension already taking an ACE inhibitor and a calcium channel blocker should be offered a thiazide-like diuretic. Thiazide-like diuretics are sulfonamide-based diuretics that have similar pharmacological properties to thiazides but have been shown in recent trials to reduce systolic blood pressure more effectively than conventional thiazides, without a significant difference in side-effect profile. Indapamide is the only thiazide-like diuretic provided in the answer choices.

Bendroflumethiazide is incorrect as thiazide-like diuretics, rather than thiazide diuretics such as bendroflumethiazide now tend to be preferred as third-line antihypertensive therapy in most patients for the reasons outlined above.

Bisoprolol is incorrect. This would be an appropriate option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

Doxazosin is incorrect. Alpha blockade with doxazosin would be another option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

Spironolactone is incorrect. Aldosterone receptor antagonism with spironolactone may be offered to patients with persistent hypertension despite taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium < 4.5mmol/L, due to the propensity of spironolactone to cause hyperkalaemia.

👍 👎 Discuss (2) Improve

Next question >

Hypertension: management ★

NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

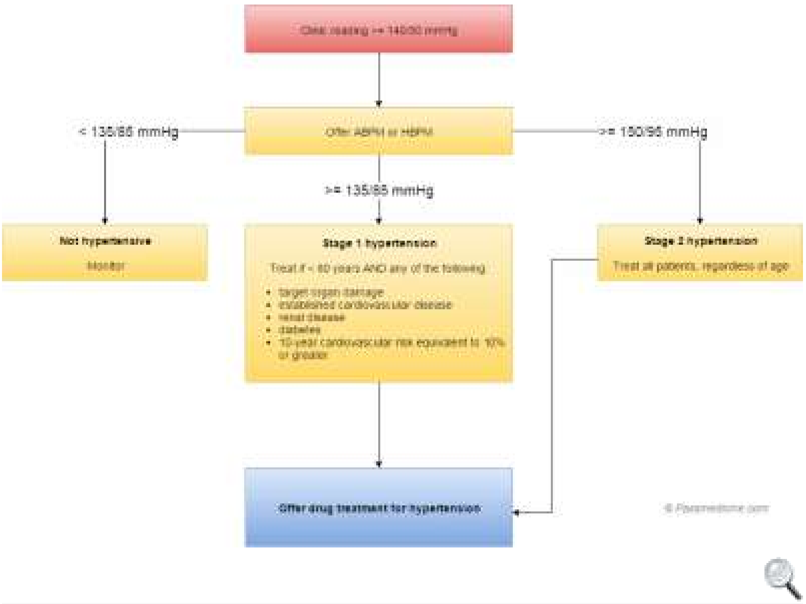
- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated

- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

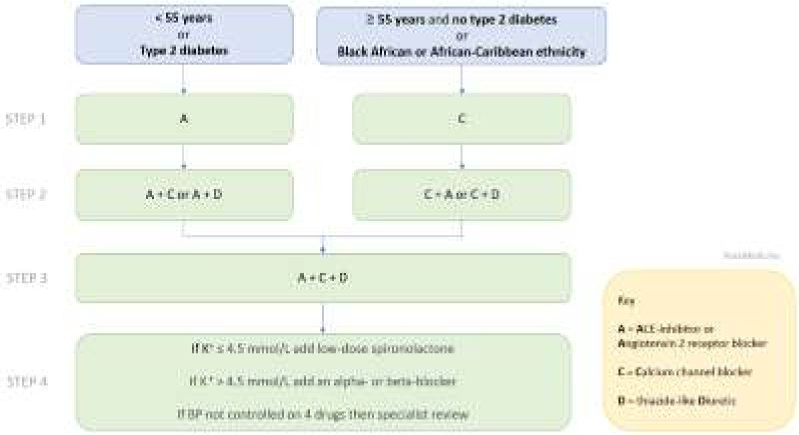
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
    - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
    - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
    - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

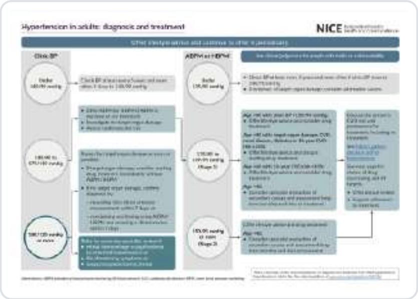
- add a third drug to make, i.e.:
    - if already taking an **(A + C)** then add a **D**
    - if already **(A + D)** then add a **C**
- **(A + C + D)**

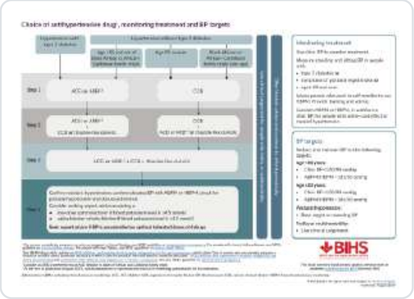
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
    - confirm elevated clinic BP with ABPM or HBPM
    - assess for postural hypotension.
    - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*





Blood pressure targets

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

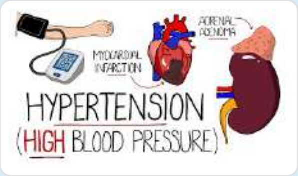
2019 Hypertension guidelines

👍 162 👎 162

Suggest link

Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **19.1%**

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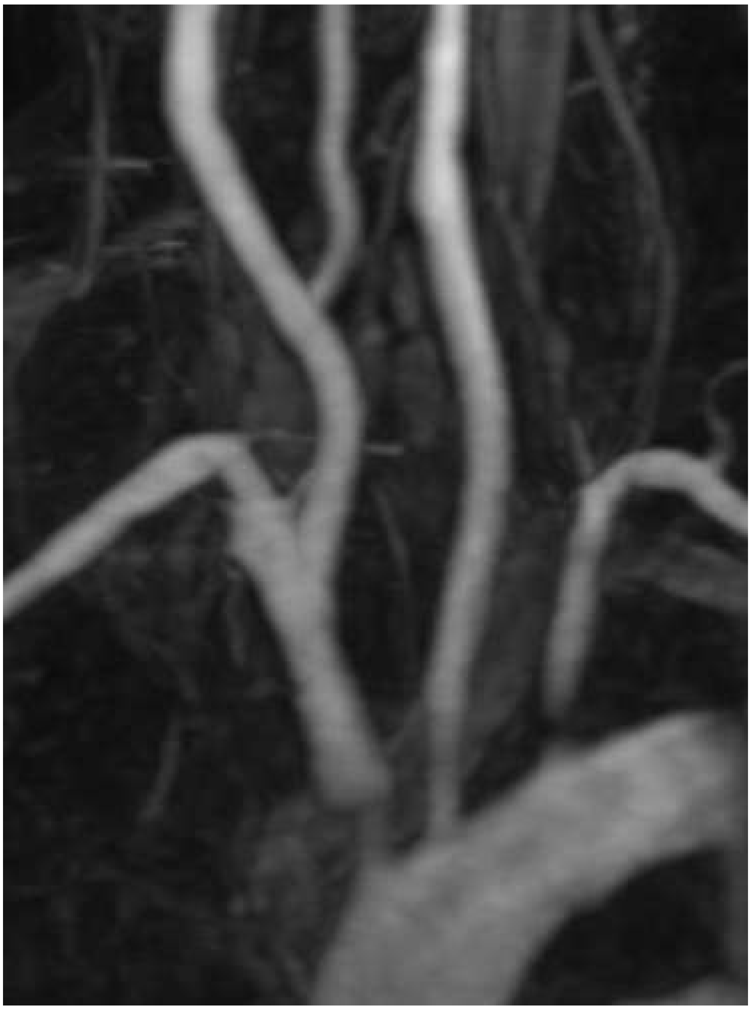
Back to top

Question 111 of 328

A 26-year-old woman is investigated for persistent malaise, fever, headaches and raised inflammatory markers. She was diagnosed with Raynaud's syndrome three years ago after complaining of cold hands. The most recent bloods in the referral letter show a CRP of 64 mg/l.

On examination blood pressure is 80/64 mmHg in the right arm and 72/54 mmHg in the left arm. The heart rate is 72/min and a diastolic murmur is noted on ascultation of the heart.

Magnetic resonance angiography is requested:



© Image used on license from Radiopaedia

What is the most appropriate maangement?

○ Plasma exchange ×

○ Rituximab

○ Prednisolone

○ Cardiothoracic surgery referral

○ Intravenous immunoglobulin

Submit answer

Reference ranges

Score: **19.1%**

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111 -

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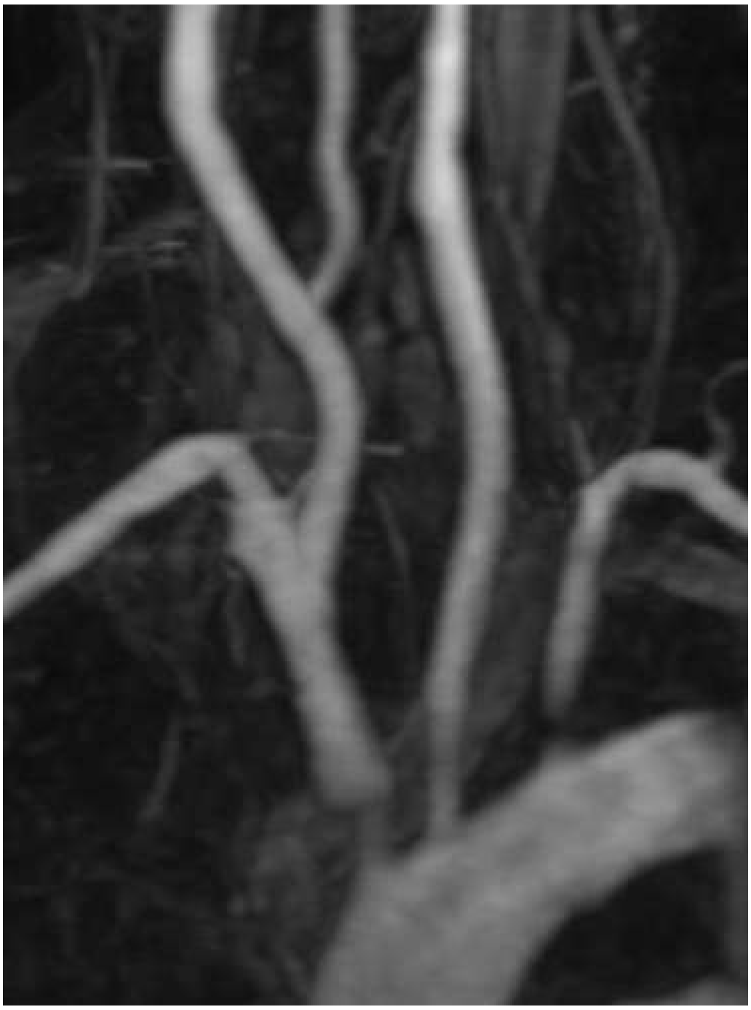


© Image used on license from Radiopaedia

What is the most appropriate maangement?

Plasma exchange 4%

Rituximab	2%
Prednisolone	56%
Cardiothoracic surgery referral	24%
Intravenous immunoglobulin	14%

The magnetic resonance angiography (MRA) demonstrates stenoses of the supra-aortic arteries, especially the brachiocephalic trunk and the left subclavian artery with near-occlusion of the left vertebral artery. This findings are consistent with a diagnosis of Takayasu's arteritis.

👍 👎 Discuss (4) Improve

Next question ›

Takayasu's arteritis

Takayasu's arteritis is a large vessel vasculitis. It typically causes occlusion of the aorta and questions commonly refer to an absent limb pulse. It is more common in younger females (e.g. 10-40 years) and Asian people.

Features
- systemic features of a vasculitis e.g. malaise, headache
- unequal blood pressure in the upper limbs
- carotid bruit and tenderness
- absent or weak peripheral pulses
- upper and lower limb claudication on exertion
- aortic regurgitation (around 20%)

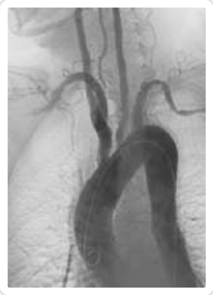
Associations
- renal artery stenosis

Investigations
- vascular imaging of the arterial tree is required to make a diagnosis of Takayasu's arteritis

    - either magnetic resonance angiography (MRA) or CT angiography (CTA)

Management

- steroids



AI + Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Media

Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology

Armando Hasudungan - YouTube 👍 4 👎 0

Takayasu arteritis
Khan Academy

Takayasu arteritis

Khan Academy - YouTube 👍 4 👎 0

Report broken media

Score: **18.9%**

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Question 112 of 328

A 34-year-old gentleman with Marfan's syndrome and known mitral regurgitation presents to the emergency department with increasing shortness of breath for three days. He can currently walk 100 metres at a time before needing to stop. He feels restless at times and has noticed his heart is racing in his chest. There is no chest pain. He is under annual surveillance with echocardiography and is currently managed on furosemide and ramipril.

On examination, his pulse is irregularly irregular at 96 beats per minute. He is alert with a clear chest and no visible JVP. He has a soft S1 with a loud grade 4 pan-systolic murmur with radiation into the axilla. He is skinny but alert.

Na^+	134 mmol/l
K^+	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	89 µmol/l

ECG	no visible P-waves, irregular narrow complex rhythm at 84 per minute
CXR	cardiomegaly with no effusions or lung shadowing
Bedside ECHO	No pericardial effusion, normal sized cardiac chambers, no regional wall motion abnormality

What is the most appropriate next action?

- ○ Increase furosemide ×
- ○ DC cardioversion ×
- ○ Bisoprolol ×
- ○ Refer to cardiothoracic surgery ×
- ○ Repeat Echocardiography ×

Submit answer

Reference ranges ⌵

Score: **18.9%**

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← Question 112 of 328 ✗ ⚐ →

A 34-year-old gentleman with Marfan's syndrome and known mitral regurgitation presents to the emergency department with increasing shortness of breath for three days. He can currently walk 100 metres at a time before needing to stop. He feels restless at times and has noticed his heart is racing in his chest. There is no chest pain. He is under annual surveillance with echocardiography and is currently managed on furosemide and ramipril.

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CXR	cardiomegaly with no effusions or lung shadowing
Bedside ECHO	No pericardial effusion, normal sized cardiac chambers, no regional wall motion abnormality

What is the most appropriate next action?

Increase furosemide	2%
DC cardioversion	6%
Bisoprolol	32%
Refer to cardiothoracic surgery	56%
Repeat Echocardiography	5%

New AF in mitral regurgitation -> refer for mitral valve replacement

Important for me Less important

This gentleman has mitral regurgitation and has developed atrial fibrillation which is a common complication following dilation of the mitral annulus. He is not in pulmonary oedema and there is no haemodynamic compromise so DC cardioversion would be inappropriate. He shows no signs of cardiac failure so diuresis with furosemide will not help. Although, he is in atrial fibrillation, his rate is slow and does not need reducing at present with a beta blocker. A repeat ECHO will be useful to confirm current heart structure. The development of atrial fibrillation is an indication to refer for mitral valve surgery as is an ejection fraction less than 60%, pulmonary hypertension or a dilated left ventricle.

👍 👎 Discuss (18) Improve

Next question >

Mitral regurgitation ★

Also known as mitral insufficiency, mitral regurgitation (MR) occurs when blood leaks back through the mitral valve on systole. It is the second most common valve disease after aortic stenosis. The mitral valve is located between the left atrium and ventricle, and regurgitation leads to a less efficient heart as less blood is pumped through the body with each contraction. This said, MR is common in otherwise healthy patients to a trivial degree and does not need treatment.

As the degree of regurgitation becomes more severe, the body's oxygen demands may exceed what the heart can supply and as a result, the myocardium can thicken over time. While this may be benign initially, patients may find themselves increasingly fatigued as a thicker myocardium becomes less efficient, and eventually go into irreversible heart failure.

Risk factors

- Female sex
- Lower body mass

- Age
- Renal dysfunction
- Prior myocardial infarction
- Prior mitral stenosis or valve prolapse
- Collagen disorders e.g. Marfan's Syndrome and Ehlers-Danlos syndrome

Causes

- Following coronary artery disease or post-MI: if the papillary muscles or chordae tendinae are affected by a cardiac insult, mitral valve disease may ensue as a result of damage to its supporting structures.
- Mitral valve prolapse: Occurs when the leaflets of the mitral valve is deformed so the valve does not close properly and allows for backflow. Most patients with this have a trivial degree of mitral regurgitation.
- Infective endocarditis: When vegetations from the organisms colonising the heart grow on the mitral valve, it is prevented from closing properly. Patients with abnormal valves are more likely to develop endocarditis as opposed to their peers.
- Rheumatic fever: While this is uncommon in developed countries, rheumatic fever can cause inflammation of the valves and therefore result in mitral regurgitation.
- Congenital

Symptoms

- Most patients with MR are asymptomatic, and patients suffering from mild to moderate MR may stay largely asymptomatic indefinitely. Symptoms tend to be due to failure of the left ventricle, arrhythmias or pulmonary hypertension. This may present as fatigue, shortness of breath and oedema.

Signs

- The murmur heard on auscultation of the chest is typically a pansystolic murmur described as 'blowing'. It is heard best at the apex and radiating into the axilla. S1 may be quiet as a result of incomplete closure of the valve. Severe MR may cause a widely split S2

Investigations

- ECG may show a broad P wave, indicative of atrial enlargement

- Cardiomegaly may be seen on chest x-ray, with an enlarged left atrium and ventricle
- Echocardiography is crucial to diagnosis and to assess severity

Treatment options

- Medical management in acute cases involves nitrates, diuretics, positive inotropes and an intra-aortic balloon pump to increase cardiac output
- If patients are in heart failure, ACE inhibitors may be considered along with beta-blockers and spironolactone
- In acute, severe regurgitation, surgery is indicated
- The evidence for repair over replacement is strong in degenerative regurgitation, and is demonstrated through lower mortality and higher survival rates
- When this is not possible, valve replacement with either an artificial valve or a pig valve is considered

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

MITRAL REGURGITATION (INSUFFICIENCY)

Mitral regurgitation

Armando Hasudungan - YouTube 👍 5 👎 0

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 1 👎 0

Mitral regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1

Report broken media

Score: **18.8%**

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Question 113 of 328

A 34-year-old male presents with increased shortness of breath on minimal exertion associated with reduced exercise tolerance. He was diagnosed with a 'heart condition' 4 years ago after three episodes of loss of consciousness. His only other past medical history is asthma, which is well controlled on a Seretide 250 inhaler. His last echocardiogram demonstrates asymmetric left ventricular and septal wall hypertrophy, associated with an increased left ventricular outflow tract gradient and systolic anterior wall motion. He was started on verapamil by his cardiologist 2 years ago but over the past 3 months, he reports a deterioration of his shortness of breath associated with increasing lower limb swelling. Which treatment is most appropriate?

- ○ Propranolol ×
- ○ Disopyramide ×
- ○ Ramipril ×
- ○ DDDR pacemaker insertion ×
- ○ Cardiac myectomy ×

Submit answer

Reference ranges

Score: **18.8%**

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113 -

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Question 113 of 328 ✗

A 34-year-old male presents with increased shortness of breath on minimal exertion associated with reduced exercise tolerance. He was diagnosed with a 'heart condition' 4 years ago after three episodes of loss of consciousness. His only other past medical history is asthma, which is well controlled on a Seretide 250 inhaler. His last echocardiogram demonstrates asymmetric left ventricular and septal wall hypertrophy, associated with an increased left ventricular outflow tract gradient and systolic anterior wall motion. He was started on verapamil by his cardiologist 2 years ago but over the past 3 months, he reports a deterioration of his shortness of breath associated with increasing lower limb swelling. Which treatment is most appropriate?

Propranolol	9%
Disopyramide	9%
Ramipril	12%
DDDR pacemaker insertion	37%
Cardiac myectomy	33%

The patient was diagnosed with echocardiogram findings associated with cardiogenic syncope in a young male, consistent with hypertrophic obstructive cardiomyopathy (HOCM). Medical treatment of HOCM consists of starting either a beta blocker, typically propranolol (but contraindicated in this case due to asthma) or a non-dihydropyridine calcium channel blocker, typically verapamil. In the case of persistent symptoms despite monotherapy, American Heart Association and European Society of Cardiology recommend consideration of disopyramide, a negative inotropic 1a anti-arrhythmic that demonstrated a significant decrease of LVOT gradients and mortality when compared to placebo. However, its significant cholinergic side effects and concerns of pro-arrhythmias, restricts its use to a second line agent. Surgical myectomy, DDDR pacemakers and alcohol ablation should be considered in patients who continue to be symptomatic despite 2 agents. ACEi should be avoided in HOCM patients with LVOT as reducing afterload can worsen the LVOT gradient.

1. Gersh BJ, Maron BJ, Bonow RO, et al. 2011 ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2011; 124:2761.
2. Sherrid MV, Barac I, McKenna WJ et al. Multicenter study of the efficacy and safety of disopyramide in obstructive hypertrophic cardiomyopathy. J Am Coll Cardiol. 2005;45(8):1251.

👍 👎 Discuss (10) Improve

Next question >

Hypertrophic obstructive cardiomyopathy: management ★

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500.

Management
- **A**miodarone
- **B**eta-blockers or verapamil for symptoms
- **C**ardioverter defibrillator
- **D**ual chamber pacemaker
- **E**ndocarditis prophylaxis*

Drugs to avoid
- nitrates
- ACE-inhibitors
- inotropes

*although see the 2008 NICE guidelines on infective endocarditis prophylaxis

Next question >

Textbooks

High-yield textbook

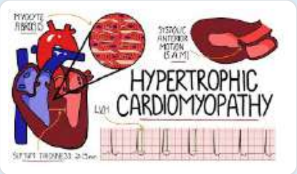
Extended textbook

Media

CARDIOLOGY
HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic obstructive cardiomyopathy

Osmosis - YouTube 👍 8 👎 0


SYSTOLIC ANTERIOR MOTION (SAM)
HYPERTROPHIC CARDIOMYOPATHY
LVH

Hypertrophic Cardiomyopathy Made Easy

Rhesus Medicine - YouTube 👍 1 👎 0

Hypertrophic cardiomyopathy

Osmosis - YouTube 👍 3 👎 4

Report broken media

Score: **18.6%**

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Back to top

A 43-year-old male presents with central chest pain that spread into his jaw and arm. He is nauseous and sweaty and arrives to the emergency department. He is immediately given aspirin and glyceryl trinitrate spray. On examination, he has normal heart sounds and a clear chest. However, he is pale and clammy

Hb	145 g/l
Platelets	400 * 10^9/l
WBC	8.6 * 10^9/l

Na$^+$	135 mmol/l
K$^+$	4.3 mmol/l
Urea	4.5 mmol/l
Creatinine	67 µmol/l

Troponin	957 ng/ml (normal range <20)
ECG	Biphasic T waves in V2-V3

What is the likely explanation of the ECG?

- ○ Normal variant
- ○ Brugada Syndrome
- ○ Wellens Syndrome
- ○ Hypertrophic cardiomyopathy
- ○ Takotsubo cardiomyopathy

Submit answer

Reference ranges

Score: **18.6%**

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Na^+	135 mmol/l
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Urea	4.5 mmol/l
Creatinine	67 μmol/l

Troponin	957 ng/ml (normal range <20)
ECG	Biphasic T waves in V2-V3

What is the likely explanation of the ECG?

Normal variant	2%
Brugada Syndrome	9%
Wellens Syndrome	80%
Hypertrophic cardiomyopathy	3%
Takotsubo cardiomyopathy	5%

Deeply inverted or biphasic T waves in V2-V3 suggests Wellen's syndrome and high risk of critical LAD stenosis

Important for me Less important

This gentleman has clear acute coronary syndrome. The ECG findings are unique to Wellens Syndrome in which there is a critical stenosis of the left anterior descending artery. These patients are at risk of imminent occlusion of this artery and should be admitted for coronary angiography and further intervention. Takotsubo cardiomyopathy has ST elevation in the apical leads (V5, V6). Hypertrophic cardiomyopathy typically has T wave inversion in the anteroseptal leads. Brugada has ST elevation and T wave inversion in V1-V2. Biphasic T-waves are not a normal variant.

👍 👎 Discuss (11) Improve

Next question ›

Acute coronary syndrome: clinical features ★

Features of acute coronary syndrome (ACS) include:

- chest pain
    - classically on the left side of the chest
    - may radiate to the left arm or neck
    - this may not always be present. Being elderly, diabetic or female makes an atypical presentation more likely
- dyspnoea
- nausea and vomiting
- sweating
- palpitations

AI + Q 123

Next question ›

B *I*

Textbooks

High-yield textbook

Extended textbook

Score: **18.4%**

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Question 115 of 328

A 65-year-old man presents with loss of consciousness. He has a past medical history of two myocardial infarctions, one of which was four weeks ago. His wife states that he felt dizzy, complained of his vision going dark, and then fell to the floor.

An ECG shows ST elevation in V1-V4 without reciprocal depression.

What is the most likely diagnosis?

- ○ Thromboembolic stroke ×
- ○ Ventricular tachycardia ×
- ○ Anterior STEMI ×
- ○ Subarachnoid haemorrhage ×
- ○ Subdural haematoma ×

Submit answer

Reference ranges

Score: **18.4%**

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115 -

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A 65-year-old man presents with loss of consciousness. He has a past medical history of two myocardial infarctions, one of which was four weeks ago. His wife states that he felt dizzy, complained of his vision going dark, and then fell to the floor.

An ECG shows ST elevation in V1-V4 without reciprocal depression.

What is the most likely diagnosis?

Thromboembolic stroke	23%
Ventricular tachycardia	23%
Anterior STEMI	23%
Subarachnoid haemorrhage	28%
Subdural haematoma	3%

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thrombo-embolisms.

Important for me   Less important

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is very suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thromboembolisms, both of which are high up the differential in this case. The patient has presented with classic features of syncope, which makes an arrhythmia much more likely than an embolic stroke which rarely presents with syncope.

Subarachnoid haemorrhage can present with both loss of consciousness and ST elevation, and would be an important differential to consider, albeit less likely.

👍 👎 Discuss (16) Improve

Next question >

Myocardial infarction: complications

Patients are at risk of a number of immediate, early and late complications following a myocardial infarction (MI).

Cardiac arrest

This most commonly occurs due to patients developing ventricular fibrillation and is the most common cause of death following a MI. Patients are managed as per the ALS protocol with defibrillation.

Cardiogenic shock

If a large part of the ventricular myocardium is damaged in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock. This is difficult to treat. Other causes of cardiogenic shock include the 'mechanical' complications such as left ventricular free wall rupture as listed below. Patients may require inotropic support and/or an intra-aortic balloon pump.

Chronic heart failure

As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop diuretics such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias

Ventricular fibrillation, as mentioned above, is the most common cause of death following a MI. Other common arrhythmias including ventricular tachycardia.

Bradyarrhythmias

Atrioventricular block is more common following inferior myocardial infarctions.

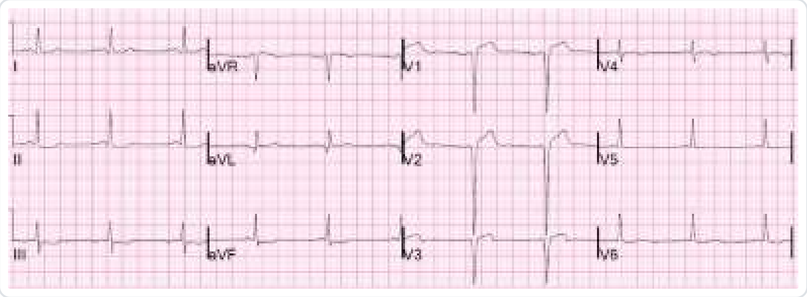
Pericarditis

Pericarditis in the first 48 hours following a transmural MI is common (c. 10% of patients). The pain is typical for pericarditis (worse on lying flat etc), a pericardial rub may be heard and a pericardial effusion may be demonstrated with an echocardiogram.

Dressler's syndrome tends to occur around 2-6 weeks following a MI. The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers. It is characterised by a combination of fever, pleuritic pain, pericardial effusion and a raised ESR. It is treated with NSAIDs.

Left ventricular aneurysm

The ischaemic damage sustained may weaken the myocardium resulting in aneurysm formation. This is typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated.



Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent pericardiocentesis and thoracotomy are required.

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. Acute hypotension and pulmonary oedema may occur. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 36 👎 22

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Score: **19.1%**

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115	✓

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Question 116 of 328

A 36 year old woman presents to the medical assessment unit with a 2 day history of pleuritic right sided chest pain, and shortness of breath. She is 38 weeks pregnant with her first child, her pregnancy so far had been uncomplicated.

On examination, she is slightly tachypnoeic, with no added sounds on chest auscultation.
There is no clinical evidence of a deep vein thrombosis. Her oxygen saturations are 95% on room air, heart rate 98/min.

What is the next appropriate investigation?

- ○ CTPA
- ○ D Dimer
- ○ Chest x-ray
- ○ V/Q scan
- ○ Bilateral leg dopplers

Submit answer

Reference ranges

Score: **19.1%**

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A 36 year old woman presents to the medical assessment unit with a 2 day history of pleuritic right sided chest pain, and shortness of breath. She is 38 weeks pregnant with her first child, her pregnancy so far had been uncomplicated.

On examination, she is slightly tachypnoeic, with no added sounds on chest auscultation.
There is no clinical evidence of a deep vein thrombosis. Her oxygen saturations are 95% on room air, heart rate 98/min.

What is the next appropriate investigation?

CTPA	3%
D Dimer	9%
Chest x-ray	65%
V/Q scan	12%
Bilateral leg dopplers	11%

Venous thromboembolic event (VTE) is an important cause of maternal death in the UK. Pregnant women are 10x more likely to have a VTE than non-pregnant women of the same age.

Where there is suspicion of a pulmonary embolism (PE) , a chest x-ray should be performed initially. If this does not explain the patient's symptoms, compression duplex dopplers should be performed. A diagnosis of deep vein thrombosis (DVT) can indirectly confirm a PE, reducing radiation doses for the mother.

If both tests are negative, and the clinical suspicion of PE remains high, further imaging should be organised; either CTPA or ventilation-perfusion scan (V/Q scan). British thoracic society guidelines (BTS) recommend a CTPA in non-pregnant women.

The decision as to which scan to perform should be ideally taken with the input from the patient. V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA but carries a lower risk of maternal breast cancer. The ventilation component of the V/Q scan may be able to be omited, reducing the radiation dose.

A D Dimer would not assist in the diagnosis, as it may be raised anyway in pregnancy due to disturbances in the coagulation system.

Guidelines: RCOG- The acute management of thrombosis and embolism during pregnancy and the puerperium.

👍 | 👎 | Discuss (5) | Improve

Next question >

Pregnancy: DVT/PE investigation ★

Guidelines were updated in 2015 by the Royal College of Obstetricians.

For patients with a suspected deep vein thrombosis (DVT):

- Compression duplex ultrasound should be undertaken where there is clinical suspicion of DVT

For patients with a suspected pulmonary embolism (PE):

- ECG and chest x-ray should be performed in all patients
- In women who also have symptoms and signs of DVT, compression duplex ultrasound should be performed. If compression ultrasonography confirms the presence of DVT, no further investigation is necessary and treatment for VTE should continue
- the decision to perform a V/Q or CTPA should be taken at a local level after discussion with the patient and radiologist

Comparing CTPA to V/Q scanning in pregnancy

CTPA	V/Q scanning
CTPA slightly increases the lifetime risk of **maternal breast cancer** (increased by up to 13.6%, background risk of 1/200 for study population). Pregnancy makes breast tissue particularly sensitive to the effects of radiation	V/Q scanning carries a slightly increased risk of **childhood cancer** compared with CTPA (1/50,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it often raised in pregnancy.

+ Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

RCOG 👍 2 👎 0

Thromboembolic disease in pregnancy and the puerperium: acute management

Suggest link Report broken link

Score: **19%**

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115 ✓
116 ✗

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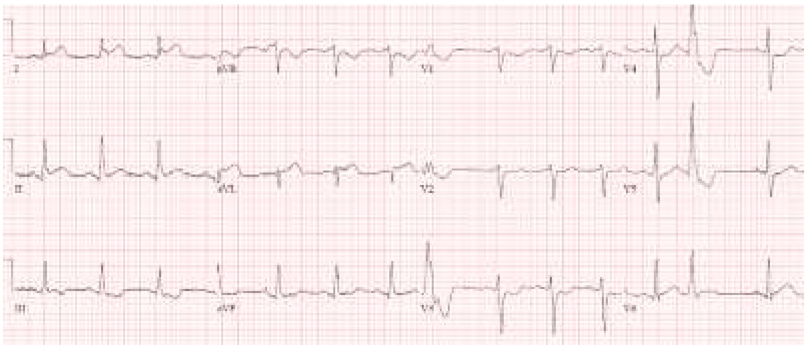
A 50-year-old woman with a past medical history of type 1 diabetes, angina, and hypertension presents with vomiting and epigastric discomfort that came on suddenly earlier this morning.

Her blood glucose level is 10.4 mmol/L and her urine dip is '+' for ketones.

Observations are as follows:

- Respiratory rate: 19/min
- Oxygen saturations: 96% on room air
- Heart rate 96bpm
- Blood pressure 105/67 mmHg

Her blood results are awaited, and her ECG is shown below.



© Image used on license from Dr Smith, University of Minnesota

What diagnostic option is most likely to confirm the diagnosis?

○ Abdominal x-ray ×

○ CT scan of the abdomen ×

○ Repeat ECG with posterior chest leads ×

○ Serum cortisol ×

○ Venous blood gas ×

Submit answer

Reference ranges ⌄

Score: **19%**

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Question 117 of 328 ✓

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- Heart rate 96bpm
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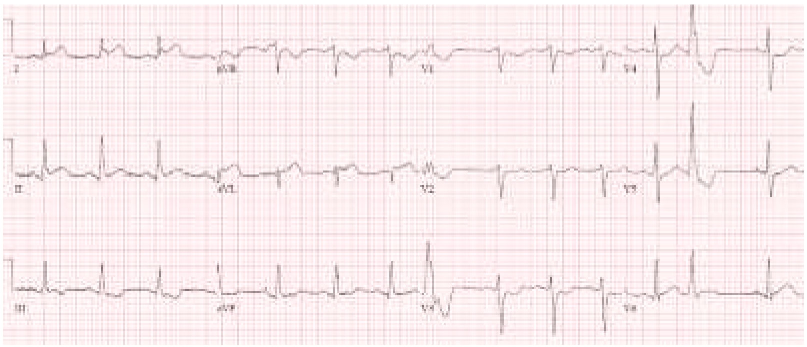
Her blood results are awaited, and her ECG is shown below.



© Image used on license from Dr Smith, University of Minnesota

What diagnostic option is most likely to confirm the diagnosis?

Abdominal x-ray	1%
CT scan of the abdomen	7%
Repeat ECG with posterior chest leads	71%
Serum cortisol	2%
Venous blood gas	19%

The ECG here shows sinus rhythm with a rate of roughly 85bpm. There is ST elevation in leads I, aVL, and v6. There is ST depression in lead v3 and v4. Conduction appears to be normal. There are two premature ventricular complexes (PVCs).

The above findings can be explained by an acute coronary syndrome involving the posterior and lateral coronary territories. Posterior chest leads would help to confirm a posterior myocardial infarction, so **repeat ECG with posterior chest leads** is the correct answer.

A **venous blood gas** would be the option of choice if aiming to confirm diabetic ketoacidosis, but given the blood glucose is <11.0 mmol/L and ketonuria is less than 2+, this diagnosis is less likely.

CT scan of the abdomen would be correct if her symptoms were thought to be coming from an intraabdominal cause, but given the ECG evidence of an acute coronary syndrome which would explain her epigastric discomfort and vomiting, this seems less likely.

Serum cortisol would be useful in confirming a diagnosis of adrenal crisis, which could present as epigastric discomfort, hypotension and vomiting. The ECG findings make the diagnosis of acute coronary syndrome more likely.

Abdominal x-ray would be useful in looking for evidence to suggest bowel obstruction or perforation. Obstruction would not explain the territorial ST-segment changes and so is not correct in this instance.

Discuss (3) Improve

Next question

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	**Coronary artery**
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

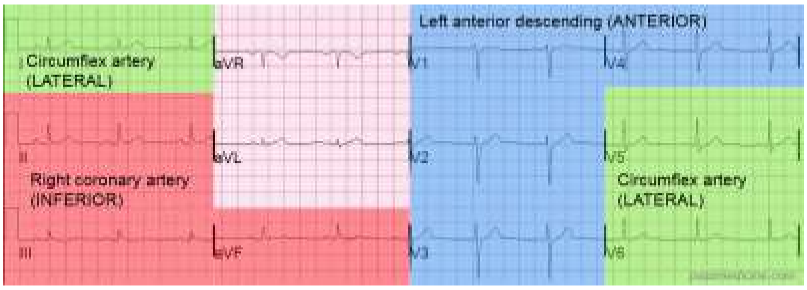

Left anterior descending (ANTERIOR)
Circumflex artery (LATERAL)
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 24 👎 40

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 14 👎 23

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **19.7%**

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117 ✓

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Question 118 of 328

A 60-year-old woman has a heart rate of 180bpm on the medical admission unit. An ECG performed demonstrates a regular narrow-complex tachycardia. The patient complains of palpitations but no chest pain and blood pressure remains 140/95. Initial vagal maneuvers are performed including carotid massage with no success. Intravenous adenosine 6mg is given followed by a further two doses of adenosine 12mg. Unfortunately, this failed to cardiovert or reveal an interpretable underlying rhythm.
What is the next management step?

- ○ Further adenosine ×
- ○ Synchronised DC cardioversion ×
- ○ Verapamil ×
- ○ Digoxin ×
- ○ Amiodarone ×

Submit answer

Reference ranges

Score: **19.7%**

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116 ✗

117 ✓

118 -

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A 60-year-old woman has a heart rate of 180bpm on the medical admission unit. An ECG performed demonstrates a regular narrow-complex tachycardia. The patient complains of palpitations but no chest pain and blood pressure remains 140/95. Initial vagal maneuvers are performed including carotid massage with no success. Intravenous adenosine 6mg is given followed by a further two doses of adenosine 12mg. Unfortunately, this failed to cardiovert or reveal an interpretable underlying rhythm.

What is the next management step?

	Further adenosine	14%
🟥	Synchronised DC cardioversion	34%
🟩	Verapamil	38%
	Digoxin	1%
	Amiodarone	14%

The resuscitation council recommends:

Adenosine 6mg for supraventricular tachycardias followed by a further two doses of adenosine 12mg if no response. If adenosine is contra-indicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating atrial flutter, consider giving a calcium-channel blocker, for example intravenous verapamil 2.5 - 5mg over 2 min.

The combination of vagal maneuvers and 6mg/12mg/12mg will terminate most regular narrow-complex tachycardias. Further adenosine is not thought to be of benefit.

Digoxin and amiodarone are used in the treatment of atrial fibrillation but not indicated in regular narrow-complex tachycardias. However, remember to consider alternative diagnoses such as atrial flutter/fibrillation, especially when the above rhythm fails to terminate after adenosine.

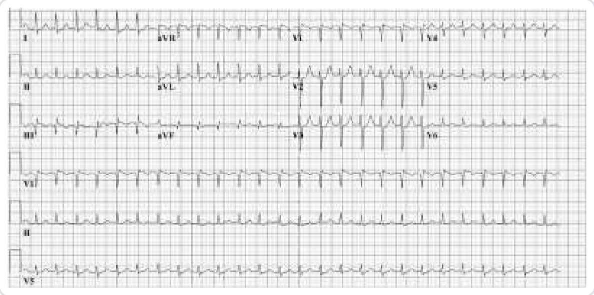
The patient is stable; with no adverse features to suggest DC cardioversion is indicated.

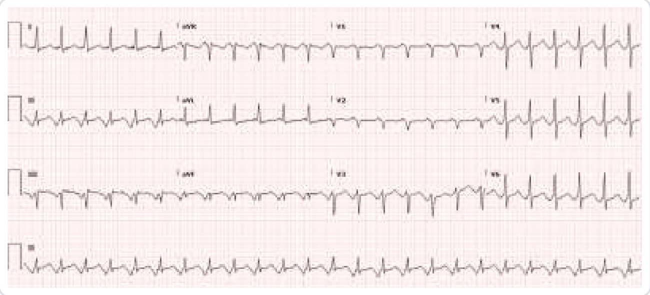
👍 👎 Discuss (11) Improve

Next question ›

Supraventricular tachycardia ★

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
    - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
    - carotid sinus massage
- intravenous adenosine
    - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg

    - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes
- beta-blockers
- radio-frequency ablation

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

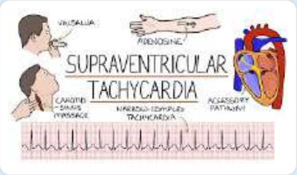
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

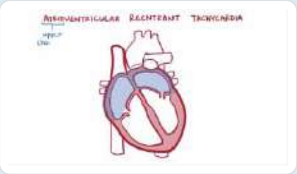
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

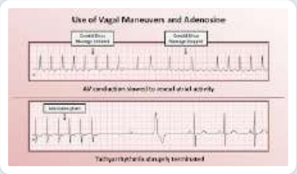
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0


Use of Vagal Maneuvers and Adenosine

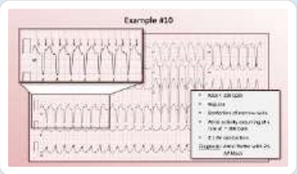
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0

AVRT & AVNRT
Khan Academy

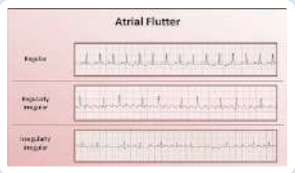
Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0



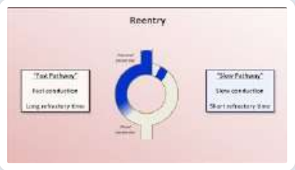
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0



Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0



Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **19.5%**

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Question 119 of 328

A 82-year-old man who is resident in a nursing home and has diabetes, hypertension and bilateral below knee amputations for peripheral vascular disease presents with a two month history of gradual deterioration. He is generally tired and has little appetite. He has lost 6 pounds since the start of the year and is not keen to leave his room anymore. He has spiked temperatures on numerous occasions and been given several course of oral antibiotics by the GP for possible urinary and lower respiratory tract infections.

On visiting him, you note a new pan systolic murmur which radiates into the axilla. He has several splinter haemorrhages and a palpable spleen. There are small haemorrhages and infarctions seen on fundoscopy. He is admitted to the emergency department?

CRP	72 mg/L
ESR	64 mm/hr
Blood cultures	*Viridans streptococci*
Transthoracic ECHO	11mm vegetation on the anterior cusp of the mitral valve

What is the likeliest cause behind this presentation?

- ○ Poor dentition
- ○ Former intravenous drug use
- ○ Childhood rheumatic fever
- ○ Senile calcification
- ○ Repeat courses of antibiotics

Submit answer

Reference ranges

Score: **19.5%**

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A 82-year-old man who is resident in a nursing home and has diabetes, hypertension and bilateral below knee amputations for peripheral vascular disease presents with a two month history of gradual deterioration. He is generally tired and has little appetite. He has lost 6 pounds since the start of the year and is not keen to leave his room anymore. He has spiked temperatures on numerous occasions and been given several course of oral antibiotics by the GP for possible urinary and lower respiratory tract infections.

On visiting him, you note a new pan systolic murmur which radiates into the axilla. He has several splinter haemorrhages and a palpable spleen. There are small haemorrhages and infarctions seen on fundoscopy. He is admitted to the emergency department?

CRP	72 mg/L
ESR	64 mm/hr
Blood cultures	*Viridans streptococci*
Transthoracic ECHO	11mm vegetation on the anterior cusp of the mitral valve

What is the likeliest cause behind this presentation?

Poor dentition	82%
Former intravenous drug use	5%
Childhood rheumatic fever	7%
Senile calcification	5%
Repeat courses of antibiotics	2%

Patients with very poor dental hygiene may develop endocarditis secondary to Viridans streptococci e.g. *Streptococcus sanguinis*

Important for me Less important

This is a case of infective endocarditis which can be acute or chronic. The chronic endocarditis tends to present over months with gradual deterioration and is most often caused by *Viridans streptococci*. This is a common oral commensal and observation of the oral cavity will likely reveal poor dental disease. This is common in people who are cared for where dental hygiene needs can be missed or difficult to achieve. Intravenous drug use is a cause commonly of *Staphylococcus aureus* endocarditis which is acute and rapidly fatal. Childhood rheumatic fever and senile calcification are rarer causes of endocarditis. Repeat courses of antibiotics causes *Clostridium difficile* colitis not endocarditis.

👍 👎 Discuss (2) Improve

Next question >

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
    - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
    - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis
    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular

    organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*
    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes
- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link

Report broken link

Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **19.3%**

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Question 120 of 328

A 78-year-old lady presents with syncope. She states that she has had several episodes of light headedness over the past couple of months. There are no obvious precipitants of the dizzy spells. She has a past medical history of angina, COPD and hypertension. Her regular medicines include GTN, aspirin, ramipril and furosemide.

ECG results are as follows:

Rate	68 beats per minute
Rhythm	Sinus rhythm
PR interval	220 ms
QRS duration	130 ms
QRS morphology	Right bundle branch block
Cardiac axis	Left axis deviation

What investigation will you order?

- ○ 7-day ECG Holter monitor ×
- ○ Transthoracic echocardiogram ×
- ○ Transesophageal echocardiogram ×
- ○ Brain natriuretic peptide (BNP) ×
- ○ Tilt-table test ×

Submit answer

Reference ranges

Score: **19.3%**

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118 ✗

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120 -

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Question 120 of 328 ✗

A 78-year-old lady presents with syncope. She states that she has had several episodes of light headedness over the past couple of months. There are no obvious precipitants of the dizzy spells. She has a past medical history of angina, COPD and hypertension. Her regular medicines include GTN, aspirin, ramipril and furosemide.

ECG results are as follows:

Rate	68 beats per minute
Rhythm	Sinus rhythm
PR interval	220 ms
QRS duration	130 ms
QRS morphology	Right bundle branch block
Cardiac axis	Left axis deviation

What investigation will you order?

7-day ECG Holter monitor	45%
Transthoracic echocardiogram	34%
Transesophageal echocardiogram	6%
Brain natriuretic peptide (BNP)	1%
Tilt-table test	14%

Patients who present with syncope and have an ECG showing incomplete trifascicular block need a Holter test to assess for episodes of complete heart block

Important for me Less important

The right bundle branch block (RBBB), left axis deviation (LAD) and prolonged PR interval are suggestive of incomplete trifascicular block.

These patients require a 7-day ECG Holter monitor to assess for episodes of complete (third degree) hear block. It is likely that a permanent cardiac pacemaker will be required to prevent further syncopal episodes.

A tilt-table test is useful for investigating vasovagal syncope.

The patient may also have underlying structural heart disease so a transthoracic echo would also be prudent to perform. However, the most immediate concern is of arrhythmia, making a 7 day holter the better answer.

👍 👎 Discuss (18) Improve

Next question >

Complete heart block ★

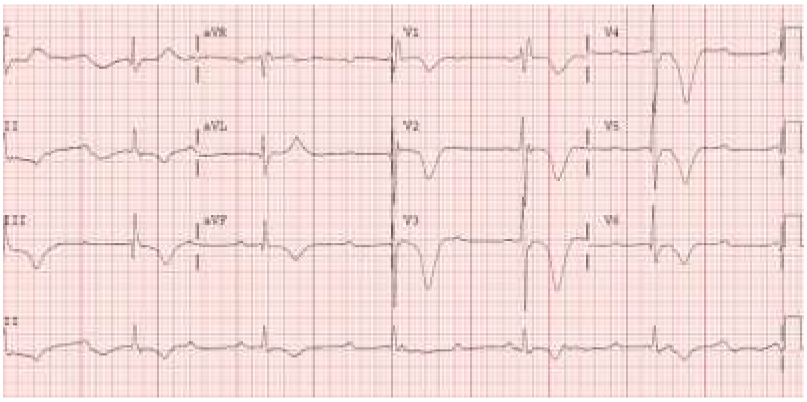
Features
- syncope
- heart failure
- regular bradycardia (30-50 bpm)
- wide pulse pressure
- JVP: cannon waves in neck
- variable intensity of S1

Types of heart block

First degree heart block
- PR interval > 0.2 seconds

Second degree heart block
- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex

Third degree (complete) heart block
- there is no association between the P waves and QRS complexes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ECG showing third degree (complete) heart block

123

Next question >

Textbooks

High-yield textbook

Extended textbook

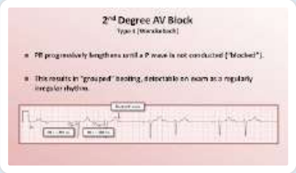
Links

ECGPedia 13 10

AV conduction problems

Suggest link Report broken link

Media

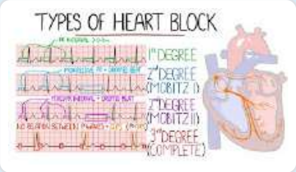


2nd Degree AV Block

- PR progressively lengthens until a P wave is not conducted ("blocked").
- This results in "grouped" beating, detectable on exam as a regularly irregular rhythm.

AV block

Strong Medicine - YouTube 👍 2 👎 0


TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Atrioventricular block

Osmosis - YouTube 👍 0 👎 1

Report broken media

Score: **19.2%**

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119 ✗

120 ✗

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Question 121 of 328

An 18-year-old man attends the clinic with blurred vision. This has progressively worsened over the past few years. He has a past medical history of recurrent deep vein thromboses and mild learning difficulty. On examination, you note an increased arm span to body height ratio, and the presence of scoliosis. Ophthalmological assessment reveals a downward lens dislocation.

What is the most likely diagnosis?

- ○ Ectopia lentis syndrome
- ○ Ehlers danlos syndrome
- ○ Homocystinuria
- ○ Marfan's syndrome
- ○ Multiple endocrine neoplasia type 2B

Submit answer

Reference ranges

Score: **19.2%**

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119 ✗

120 ✗

121 -

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Question 121 of 328 ×

An 18-year-old man attends the clinic with blurred vision. This has progressively worsened over the past few years. He has a past medical history of recurrent deep vein thromboses and mild learning difficulty. On examination, you note an increased arm span to body height ratio, and the presence of scoliosis. Ophthalmological assessment reveals a downward lens dislocation.

What is the most likely diagnosis?

Ectopia lentis syndrome	2%
Ehlers danlos syndrome	6%
Homocystinuria	60%
Marfan's syndrome	31%
Multiple endocrine neoplasia type 2B	1%

Tall, long fingered, downward lens dislocation, learning difficulties, DVT - homocystinuria

Important for me Less important

The patient has several clinical features of a Marfanoid body habitus. The differential for a Marfanoid body habitus is wide and includes Marfan's syndrome and homocystinuria.

Homocystinuria is correct. The patient has several clinical features associated with homocystinuria (e.g. Marfanoid body habitus, recurrent DVTs, learning disability and ectopia lentis). It is important to remember that the ectopia lentis in homocystinuria is inferonasal, in contrast to the superior-temporally dislocation associated with Marfan's syndrome.

Ectopia lentis syndrome is incorrect. Ectopia lentis syndrome is an inherited connective tissue condition that shares some of the features of Marfan's syndrome - particularly lens dislocation of the eye. However, patients with ectopia lentis syndrome do not have any vascular complications associated with Marfan's syndrome. Patients with ectopia lentis syndrome do not have any of the other features demonstrated in this case.

Ehlers danlos syndrome is incorrect. The Ehlers-Danlos syndromes (EDS) are generally characterised by joint hypermobility, joint instability and dislocations, scoliosis, and other joint deformities. In the rarer types of EDS, there is also a weakness of specific tissues that can lead, for example, to major gum and dental disease, eye disease, cardiac valve and aortic root disorders, and life-threatening abdominal organ, uterine, or blood vessel rupture. Thrombotic complications and lens dislocation are rare in this condition making it a less likely diagnosis.

Marfan's syndrome is incorrect. Although the patient has a Marfanoid body habitus, the presence of downward lens dislocation, learning difficulties, and recurrent DVTs favour the diagnosis of homocystinuria.

Multiple endocrine neoplasia type 2B is incorrect. This is a rare, genetic disorder that affects the endocrine glands and causes medullary thyroid cancer, pheochromocytoma, and parathyroid gland cancer. Although it can be associated with a Marfanoid body habitus, there are no other clinical features to suggest that this is the diagnosis.

👍 👎 Discuss (2) Improve

Next question >

Homocystinuria ★

Homocystinuria is a rare autosomal recessive disease caused by a deficiency of cystathionine beta synthase. This results in severe elevations in plasma and urine homocysteine concentrations.

Features
- often patients have fine, fair hair
- musculoskeletal
  - Marfanoid body habitus: arachnodactyly etc
  - osteoporosis
  - kyphosis
- neurological: may have learning difficulties, seizures

- ocular
    - downwards (inferonasal) dislocation of lens
    - severe myopia
- increased risk of arterial and venous thromboembolism
- also malar flush, livedo reticularis

Investigations

- increased homocysteine levels in serum and urine
- cyanide-nitroprusside test: also positive in cystinuria

Treatment is vitamin B6 (pyridoxine) supplements.

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 6 👎 2

Homocystinuria review

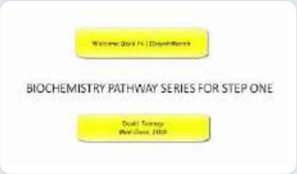
Suggest link Report broken link

Media

HOMOCYSTINURIA
Osmosis.org

Homocystinuria

Osmosis - YouTube 👍 4 👎 0


BIOCHEMISTRY PATHWAY SERIES FOR STEP ONE

Homocysteine Metabolism

12DaysinMarch - YouTube 👍 3 👎 1

Report broken media

Score: **19%**

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Question 122 of 328

A 67-year-old with a history of ischaemic heart disease (primary percutaneous intervention for a STEMI three years ago) is admitted with a pyrexia of unknown origin. On examination his pulse is 96/min, temperature 38.2ºC and blood pressure 104/66 mmHg. A systolic murmur is noted but auscultation of the chest is unremarkable. His post-myocardial infarction echocardiogram three years ago showed no valvular disease. Chest x-ray is normal and urine dipstick shows blood ++. A petechial rash is noted on his hands and legs. A presumptive diagnosis of infective endocarditis is made and empirical treatment with IV amoxicillin and gentamicin given. Two days later blood cultures show a coagulase-negative staphylococcus. What is the most appropriate action with respect to antibiotic therapy?

- ○ Switch to flucloxacillin
- ○ Switch to flucloxacillin + vancomycin + rifampicin
- ○ Make no changes to treatment
- ○ Switch to flucloxacillin + vancomycin
- ○ Switch to flucloxacillin + rifampicin

Submit answer

Reference ranges

Score: **19%**

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A 67-year-old with a history of ischaemic heart disease (primary percutaneous intervention for a STEMI three years ago) is admitted with a pyrexia of unknown origin. On examination his pulse is 96/min, temperature 38.2ºC and blood pressure 104/66 mmHg. A systolic murmur is noted but auscultation of the chest is unremarkable. His post-myocardial infarction echocardiogram three years ago showed no valvular disease. Chest x-ray is normal and urine dipstick shows blood ++. A petechial rash is noted on his hands and legs. A presumptive diagnosis of infective endocarditis is made and empirical treatment with IV amoxicillin and gentamicin given. Two days later blood cultures show a coagulase-negative staphylococcus. What is the most appropriate action with respect to antibiotic therapy?

Switch to flucloxacillin ✓	59%
Switch to flucloxacillin + vancomycin + rifampicin ✗	8%
Make no changes to treatment	13%
Switch to flucloxacillin + vancomycin	13%
Switch to flucloxacillin + rifampicin	6%

The BNF now recommend flucloxacillin monotherapy for native-valve endocarditis caused by staphylococci.

Discuss (8) Improve

Next question >

Infective endocarditis: prognosis and management ★

Poor prognostic factors

- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism
- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin If prosthetic valve • vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA • vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA • vancomycin + rifampicin + low-dose gentamicin

Scenario	Suggested antibiotic therapy
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic • vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic • vancomycin + low-dose gentamicin

Indications for surgery
- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

[Infective endocarditis guidelines]

Suggest link

Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **18.9%**

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Question 123 of 328

A 49-year-old Afro-Carribean man is diagnosed with hypertension. He has no significant past medical history and remains asymptomatic. His 24 hour blood pressure monitoring shows an average of 165/97mm Hg. Which of the following drugs should be used initially to lower his blood pressure?

○ Ramipril ×
○ Bisoprolol ×
○ Amlodipine ×
○ Indapamide ×
○ Furosemide ×

Submit answer

Reference ranges

Score: **18.9%**

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Question 123 of 328

A 49-year-old Afro-Carribean man is diagnosed with hypertension. He has no significant past medical history and remains asymptomatic. His 24 hour blood pressure monitoring shows an average of 165/97mm Hg. Which of the following drugs should be used initially to lower his blood pressure?

Ramipril	6%
Bisoprolol	0%
Amlodipine	91%
Indapamide	2%
Furosemide	0%

Newly diagnosed patient of black African or African-Caribbean origin with hypertension - add a calcium channel blocker

Important for me Less important

This patient has stage two hypertension. Because he is of Afro-Caribbean origin, the first line drug is a calcium channel blocker, not an angiotensin converting enzyme inhibitor. Diuretics and beta-blockers are not first line management for hypertension.

Discuss (5) Improve

Next question

Hypertension: management

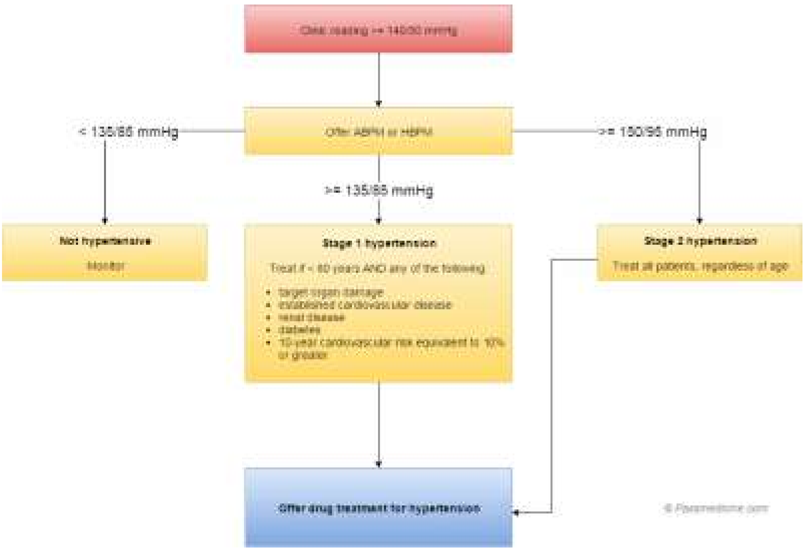
NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

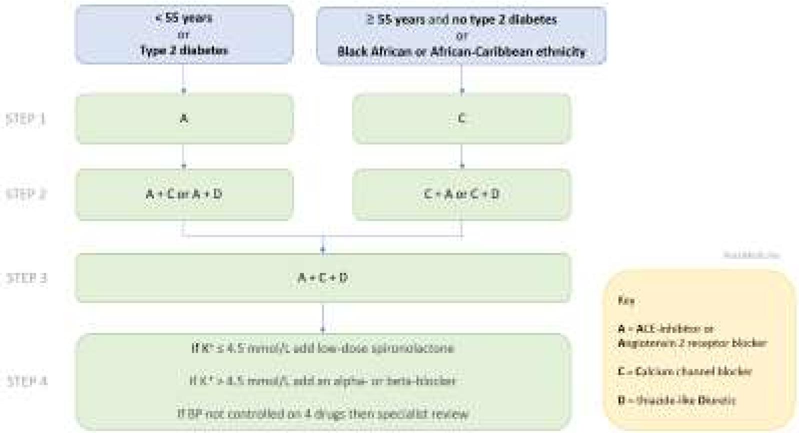
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
  - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

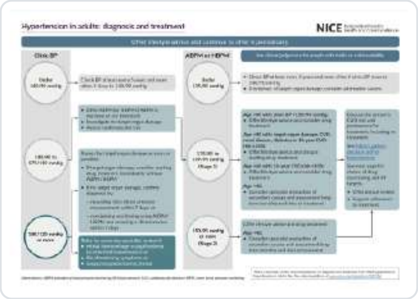
- add a third drug to make, i.e.:
  - if already taking an **(A + C)** then add a **D**
  - if already **(A + D)** then add a **C**
- **(A + C + D)**

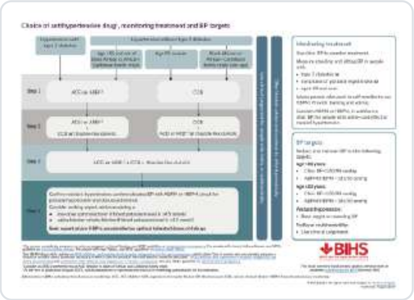
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
    - confirm elevated clinic BP with ABPM or HBPM
    - assess for postural hypotension.
    - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*





Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg

	Clinic BP	ABPM / HBPM
Age > 80 years	150/90 mmHg	145/85 mmHg

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

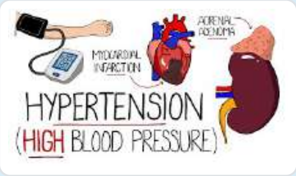
👍 162 👎 162

2019 Hypertension guidelines

Suggest link

Report broken link

Media


MYOCARDIAL INFARCTION
ADRENAL ADENOMA
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **18.7%**

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123 ✗

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A 73-year-old man with a history of severe biventricular heart failure comes to the clinic for review. He can only walk around 100 metres to the bus stop and climbs the stairs once per day only. Medication includes ramipril 10mg, furosemide 80mg, bisoprolol 10mg, spironolactone 25mg, aspirin and atorvastatin. On examination his blood pressure is 123/82 mmHg, pulse is 85 beats per minute at rest. He has bilateral basal crackles on auscultation of the chest and mild pitting oedema of both ankles.

Investigations

Hb	122 g/l	Na$^+$	134 mmol/l	Bilirubin	11 µmol/l
Platelets	190 * 10^9/l	K$^+$	5.0 mmol/l	ALP	105 u/l
WBC	7.4 * 10^9/l	Urea	11.2 mmol/l	ALT	33 u/l
Neuts	4.5 * 10^9/l	Creatinine	142 µmol/l	γGT	56 u/l
Lymphs	1.2 * 10^9/l			Albumin	38 g/l
Eosin	0.5 * 10^9/l				

Chest x-ray: Cardiomegaly, bilateral upper lobe diversion

ECG: Left bundle branch block, ventricular rate 85

ECHO: Ejection fraction 31%

Which of the following is the most appropriate next step with regards to his heart failure medication?

○ Add digoxin ×

○ Add valsartan ×

○ Add ivabradine ×

○ Increase furosemide by 40mg per day ×

○ Increase spironolactone to 100mg per day ×

Submit answer

Reference ranges ⌄

Score: **18.7%**

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Back to top

Question 124 of 328 ✕

A 73-year-old man with a history of severe biventricular heart failure comes to the clinic for review. He can only walk around 100 metres to the bus stop and climbs the stairs once per day only. Medication includes ramipril 10mg, furosemide 80mg, bisoprolol 10mg, spironolactone 25mg, aspirin and atorvastatin. On examination his blood pressure is 123/82 mmHg, pulse is 85 beats per minute at rest. He has bilateral basal crackles on auscultation of the chest and mild pitting oedema of both ankles.

Investigations

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Lymphs	$1.2 * 10^9/l$			Albumin	38 g/l
Eosin	$0.5 * 10^9/l$				

Chest x-ray: Cardiomegaly, bilateral upper lobe diversion

ECG: Left bundle branch block, ventricular rate 85

ECHO: Ejection fraction 31%

Which of the following is the most appropriate next step with regards to his heart failure medication?

Add digoxin	6%
Add valsartan	5%
Add ivabradine	76%
Increase furosemide by 40mg per day	9%

Increase spironolactone to 100mg per day 5%

The correct answer is add ivabradine. This patient has uncontrolled symptoms of heart failure despite near maximal medical therapy. His heart rate is greater than 75 beats per minute at rest, according to NICE guidelines this means ivabradine is indicated as a potential intervention. The ivabradine outcome study in heart failure indicated an 18% relative risk reduction in cardiac events (mainly driven by heart failure admissions and heart failure deaths), for the intervention added in to standard of care versus standard of care alone.

http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(10)61198-1/abstract

https://www.nice.org.uk/guidance/ta267?unlid=8503236922015121485640

A large meta-analysis of use of digoxin in heart failure suggested a 14% increase in mortality associated with its' use. This has led to a reduction in use of digoxin for heart failure in recent years. With respect to increasing spironolactone or adding valsartan, these options are likely to significantly increase risk of hyperkalaemia with limited benefit in improving symptoms. Although increasing furosemide may reduce symptoms of heart failure, it may also precipitate a large rise in serum creatinine.

Discuss (6) Improve

Next question

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

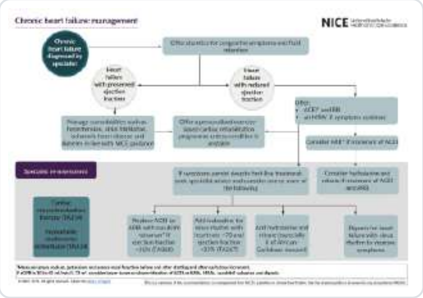
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%

    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

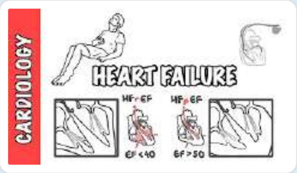
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

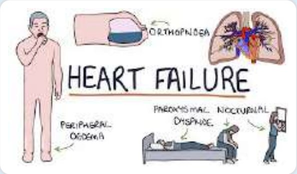
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **18.5%**

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Question 125 of 328

A 77-year-old female has been admitted electively for investigation of raised pulmonary arterial pressures (50 mmHg) detected on a transthoracic echocardiogram, which had otherwise demonstrated normal left ventricular function and chamber sizes. No valve abnormalities were identified. Her right heart catheter saturations are shown as below:

SVC	76%
IVC	74%
Right atrium (high)	73.5%
Right atrium (mid)	73%
Right atrium (low)	72.7%
Right ventricle	72%
Pulmonary artery	70.8%
Pulmonary capillary wedge	95%

What is the most likely diagnosis?

○ Normal study ×

○ Patent ductus arteriosus ×

○ Anomalous pulmonary venous drainage to SVC ×

○ Patent foramen ovale ×

○ Tricuspid regurgitation ×

Submit answer

Reference ranges

A 77-year-old female has been admitted electively for investigation of raised pulmonary arterial pressures (50 mmHg) detected on a transthoracic echocardiogram, which had otherwise demonstrated normal left ventricular function and chamber sizes. No valve abnormalities were identified. Her right heart catheter saturations are shown as below:

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IVC	74%
Right atrium (high)	73.5%
Right atrium (mid)	73%
Right atrium (low)	72.7%
Right ventricle	72%
Pulmonary artery	70.8%
Pulmonary capillary wedge	95%

What is the most likely diagnosis?

Normal study	48%
Patent ductus arteriosus	18%
Anomalous pulmonary venous drainage to SVC	18%
Patent foramen ovale	10%
Tricuspid regurgitation	6%

This is a very subtle question. When interpreting right heart catheter data, remember the saturation should decrease gradually as the venous blood reaches the pulmonary capillary wedge saturation, which should be equal to arterial blood. The second consideration for normal venous return is that oxygenation in the superior vena cava should always be lower than the inferior vena cava, due to the high oxygen demands from the brain. There are no sudden increases in oxygen saturations in this study, suggestive of a left to right shunt but the SVC saturations is markedly higher than the IVC, allowing a diagnosis of anomalous pulmonary venous drainage of more highly oxygenated blood into the SVC.

👍 👎 Discuss (8) Improve

Next question >

Cardiac catheterisation and oxygen saturation levels ★

Questions regarding cardiac catheterisation and oxygen saturation levels can seem daunting at first but a few simple rules combined with logical deduction can usually produce the answer.

Let's start with the basics:
- deoxygenated blood returns to the right side of the heart via the superior vena cava (SVC) and inferior vena cava (IVC). It has an oxygen saturation level of around **70%**. The right atrium (RA), right ventricle (RV) and pulmonary artery (PA) normally have oxygen saturation levels of around 70%
- the lungs oxygenate the blood to a level of around **98-100%**. The left atrium (LA), left ventricle (LV) and aorta should all, therefore, have oxygen saturation levels of 98-100%

The table below shows the **oxygen saturations** that would be expected in different scenarios:

Diagnosis & notes	**RA**	**RV**	**PA**	**LA**	**LV**	**Aorta**
Normal	70%	70%	70%	100%	100%	100%
Atrial septal defect (ASD) The oxygenated blood in the LA mixes with the	85%	85%	85%	100%	100%	100%

Diagnosis & notes	RA	RV	PA	LA	LV	Aorta
deoxygenated blood in the RA, resulting in intermediate levels of oxygenation from the RA onwards						
Ventricular septal defect (VSD) The oxygenated blood in the LV mixes with the deoxygenated blood in the RV, resulting in intermediate levels of oxygenation from the RV onwards. The RA blood remains deoxygenated	70%	85%	85%	100%	100%	100%
Patent ductus arteriosus (PDA) Remember, a PDA connects the higher pressure aorta with the lower pressure PA. This results in only the PA having intermediate oxygenation levels	70%	70%	85%	100%	100%	100%
VSD with Eisenmenger's	70%	70%	70%	100%	85%	85%
PDA with Eisenmenger's	70%	70%	70%	100%	100%	85%
ASD with Eisenmenger's	70%	70%	70%	85%	85%	85%

AI + Q 123

Next question ›

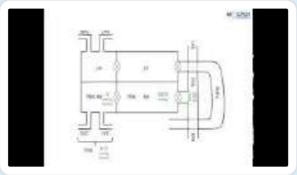
B I A

Textbooks

High-yield textbook

Extended textbook

Media



Interpreting Cardiac Catheterisation Data

MRCPCH Revision - YouTube 👍 25 👎 5

Report broken media

Score: **18.4%**

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125 ✕

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A 72-year-old gentleman is admitted with syncope and palpitations. He is known to have atrial fibrillation and is on flecainide and warfarin. His past medical history also includes benign prostatic hypertrophy and essential thrombocytosis.

His other medications include tiotropium 18 micrograms OD, tamsulosin 400 micrograms OD, ramipril 2.5mg and simvastatin 40mg. He has recently stopped a tablet due to shortness of breath but cannot remember what it was.

On arrival, he is tachycardic with a heart rate of 215 /min. He is alert. He denies chest pain but is aware of some shortness of breath. He is attached to a 3-lead cardiac monitor which shows a narrow complex tachycardia of 210-220 beats per minute. It is difficult to tell on the monitor however it appears regular. A 12-lead ECG confirms a regular narrow complex tachycardia with a ventricular rate of 215/min.

What is the most likely diagnosis?

- ○ Atrial fibrillation
- ○ Atrial flutter with 1:1 conduction
- ○ Atrial flutter with variable conduction
- ○ Atrio-ventricular re-entry tachycardia (AVRT)
- ○ Atrio-ventricular nodal re-entry tachycardia (AVNRT)

Submit answer

Reference ranges

Score: **18.4%**

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A 72-year-old gentleman is admitted with syncope and palpitations. He is known to have atrial fibrillation and is on flecainide and warfarin. His past medical history also includes benign prostatic hypertrophy and essential thrombocytosis.

His other medications include tiotropium 18 micrograms OD, tamsulosin 400 micrograms OD, ramipril 2.5mg and simvastatin 40mg. He has recently stopped a tablet due to shortness of breath but cannot remember what it was.

On arrival, he is tachycardic with a heart rate of 215 /min. He is alert. He denies chest pain but is aware of some shortness of breath. He is attached to a 3-lead cardiac monitor which shows a narrow complex tachycardia of 210-220 beats per minute. It is difficult to tell on the monitor however it appears regular. A 12-lead ECG confirms a regular narrow complex tachycardia with a ventricular rate of 215/min.

What is the most likely diagnosis?

Atrial fibrillation	2%
[✓ Atrial flutter with 1:1 conduction]	32%
Atrial flutter with variable conduction	6%
Atrio-ventricular re-entry tachycardia (AVRT)	21%
Atrio-ventricular nodal re-entry tachycardia (AVNRT)	38%

Flecainide is a pure sodium channel blocker which prolongs the cardiac action potential to slow conduction. It is recognised to increase the risk of AF transforming into flutter. Flutter with 1:1 conduction would result in a ventricular rate of 300 /min which is impossible to conduct via the AV node. However, because flecainide slows the rate of cardiac conduction it means that if a patient develops atrial flutter the atrial rate will not be 300 /min, but more likely around 200 /min which then could conduct via the AV node at a 1:1 ratio. For this reason, patients should always be co-prescribed a beta blocker to reduce risk if this happens, and you wonder if this was the tablet that had recently been stopped.

The key to this question is recognising the increased risk of atrial fibrillation transforming into atrial flutter in patients on flecainide. The regular rhythm on ECG makes atrial fibrillation and atrial flutter with variable conduction less likely. He has no specific risk factors for AVRT or AVNRT, however, his flecainide use increases the chance of atrial flutter and therefore this is the correct answer.

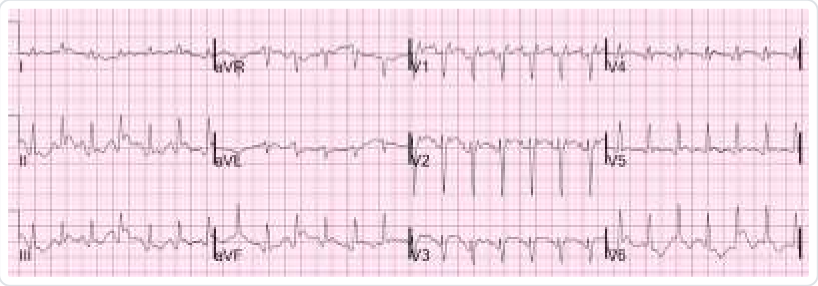
👍 👎 Discuss (10) Improve

Next question >

Atrial flutter

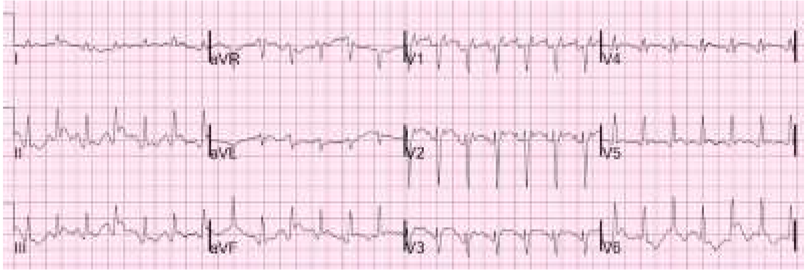
Atrial flutter is a form of supraventricular tachycardia characterised by a succession of rapid atrial depolarisation waves.

ECG findings
- 'sawtooth' appearance
- as the underlying atrial rate is often around 300/min the ventricular or heart rate is dependent on the degree of AV block. For example if there is 2:1 block the ventricular rate will be 150/min
- flutter waves may be visible following carotid sinus massage or adenosine



Management
- is similar to that of atrial fibrillation although medication may be less effective
- atrial flutter is more sensitive to cardioversion however so lower energy levels may be used

- radiofrequency ablation of the tricuspid valve isthmus is curative for most patients



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123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 3 1

Atrial Flutter

Suggest link Report broken link

Media

Atrial flutter (type 1, type 2)

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **19%**

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An 87-year-old gentleman is reviewed in the cardiology clinic. He has unstable angina with multiple confluent lesions in all three main coronary vessels and in major tributaries on angiography. The lesions are not amenable to stenting or bypass. On assessment in the clinic, the blood pressure is 104/43mmHg, heart rate is 57bpm. His ECG confirms atrial fibrillation but no new ischaemic changes. There is no clinical sign of heart failure. He is currently taking aspirin, bisoprolol, ramipril, isosorbide mononitrate, nicorandil and simvastatin. His main symptom is chest pain on minimal exertion.

Which of the following is the most appropriate drug to control this patients angina?

○ Diltiazem ×

○ Digoxin ×

○ Ivabradine ×

○ Omega 3 ×

○ Ranolazine ×

Submit answer

Reference ranges

Score: **19%**

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2 ×
3 ×

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An 87-year-old gentleman is reviewed in the cardiology clinic. He has unstable angina with multiple confluent lesions in all three main coronary vessels and in major tributaries on angiography. The lesions are not amenable to stenting or bypass. On assessment in the clinic, the blood pressure is 104/43mmHg, heart rate is 57bpm. His ECG confirms atrial fibrillation but no new ischaemic changes. There is no clinical sign of heart failure. He is currently taking aspirin, bisoprolol, ramipril, isosorbide mononitrate, nicorandil and simvastatin. His main symptom is chest pain on minimal exertion.

Which of the following is the most appropriate drug to control this patients angina?

Diltiazem	13%
Digoxin	13%
Ivabradine	10%
Omega 3	1%
Ranolazine	63%

National guidelines were published by the Scottish Intercollegiate Guideline Network (SIGN) on the management of stable angina in 2007 with updates by NICE in 2011. Stable angina is defined as chest pain of cardiac aetiology which is precipitated by exertion with a demonstrable flow limiting lesion on angiography. Many patients who experience angina will ultimately go on to have revascularisation procedures to improve or restore coronary flow which will improve their symptoms of angina, however while waiting for interventional procedures optimisation of drug therapy is the aim. Drug therapy also is considered first-line treatment for non-critical lesions.

First line therapy in the treatment of angina is beta-blockade. Beta-blockers reduce myocardial oxygen consumption by acting as a negative inotrope and also by reducing heart rate. Some patients are unable to take beta-blockers such as asthmatics, or find that side effects such as tiredness, eczema or sexual dysfunction mean they are not tolerable. In these instances, use of calcium channel blockers such as diltiazem are recommended as first-line treatment. Since both of these classes of drug cause a reduction in heart rate and blood pressure their use must be monitored carefully, especially in the elderly. Concomitant use of beta-blockers and calcium channel blockers should be avoided where possible due to the risk of high degree conduction block and cardiac arrest.

Long-acting nitrates, such as isosorbide mononitrate or dinitrate are considered second-line therapy in stable angina. They can be used as monotherapy or as an adjunct to beta-blocker or calcium channel blocker therapy. They work by causing vasodilatation; both drugs possess a nitric oxide moiety which causes endothelial relaxation in blood vessels. This causes coronary vasodilation and improved blood flow to the myocardium with improved symptoms. A consequence of vasodilatation is hypotension and this should be considered when prescribing this drug to patients.

Another second-line option drug for stable angina in those patients who cannot tolerate nitrates or they are ineffective is nicorandil. This drug has a similar ultimate effect in that it causes dilatation of peripheral and coronary vessels to reduce symptoms of angina. It works by activating potassium channels on endovascular smooth muscle cells reducing intracellular calcium and hence relaxing the blood vessel. It too can cause profound hypotension and severe headaches.

Ivabradine is a drug which can be used for both angina and heart failure. It works by reducing heart rate to reduce myocardial oxygen demand and improve diastolic function. In this case, the heart rate is adequately controlled so reduction is not necessary. Ivabradine also works by inhibiting the hearts natural pacemaker potential in the sinoatrial node, hence it only works when the patient is in sinus rhythm, unlike here.

Ranolazine is a viable option in the management of angina where other avenues have been exhausted. It works by inhibiting the delayed sodium influx channel in the myocardium, reducing the intracellular calcium concentrations in the heart muscle. This, in turn, leads to a negative inotropic effect and reduction in symptoms of angina. Ranolazine does not cause a profound hypotensive effect and is the best choice in this scenario.

Digoxin is not commented upon as a specific treatment for heart failure in NICE guidance although it does have a modest positive inotropic effect. The relative bradycardia of the patient in the vignette makes this an unwise choice here.

Omega 3 and other fatty fish oils are not recommended to be prescribed to heart failure patients in NICE guidance due to lack of evidence of any effectiveness.

👍 👎 Discuss (14) Improve

Next question >

Angina pectoris: drug management

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication
- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate

    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE 21 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

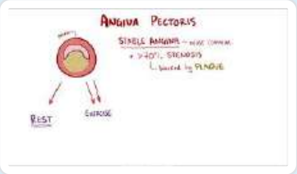
Suggest link Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **18.9%**

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A 32-year-old primigravida at 37 weeks attends the antenatal unit complaining of abdominal pain which is worse on the right side. She has also been vomiting. Her blood pressure is 148/97 mmHg. She denies any abnormal discharge and reports that fetal movements are still present. Her blood results are shown below.

Hb	93 g/l
Platelets	89 * 10^9/l
WBC	9.0 * 10^9/l
Urate	0.49 mmol/l
Bilirubin	32 µmol/l
ALP	203 u/l
ALT	190 u/l
AST	233 u/l

You are phoned for advice on the results. What is the most likely diagnosis?

○ HELLP syndrome ×

○ Obstetric cholestasis ×

○ Acute fatty liver ×

○ Hyperemesis gravidarum ×

○ Gout ×

Submit answer

Reference ranges

A 32-year-old primigravida at 37 weeks attends the antenatal unit complaining of abdominal pain which is worse on the right side. She has also been vomiting. Her blood pressure is 148/97 mmHg. She denies any abnormal discharge and reports that fetal movements are still present. Her blood results are shown below.

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Bilirubin	32 µmol/l
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AST	233 u/l

You are phoned for advice on the results. What is the most likely diagnosis?

HELLP syndrome	90%
Obstetric cholestasis	5%
Acute fatty liver	4%
Hyperemesis gravidarum	0%
Gout	0%

The most likely diagnosis here is HELLP syndrome (haemolysis, elevated liver enzymes and low platelets), a serious manifestation of pre-eclampsia. The clinical features of hypertension, vomiting and abdominal pain support the diagnosis but are not pre-requisites. The abdominal pain here may be a sign of liver inflammation and resulting stretch of the liver capsule.

Obstetric cholestasis is associated with intense pruritus and the most sensitive marker is a rise in serum bile acids. Acute fatty liver is another serious condition, which also has associations with pre-eclampsia. It would typically cause greater elevations in liver enzymes and a deep jaundice. Hyperuricaemia may be a useful marker of pre-eclampsia and does not necessarily indicate an attack of gout. Urate is thought to rise due to diminished kidney function and reduced clearance. Hyperemesis gravidarum should be a diagnosis of exclusion and would be unlikely to present for the first time this late into pregnancy.

👍 👎 Discuss (3) Improve

Next question >

Pre-eclampsia ★

Pre-eclampsia describes the emergence of high blood pressure during pregnancy that may be a precursor to a woman developing eclampsia and other complications. It is classically a triad of 3 things:

- new-onset hypertension
- proteinuria
- oedema

Definition

The current formal definition is as follows

- new-onset blood pressure ≥ 140/90 mmHg after 20 weeks of pregnancy, AND 1 or more of the following:
  - proteinuria
  - other organ involvement (see list below for examples): e.g. renal insufficiency (creatinine ≥ 90 umol/L), liver, neurological, haematological, uteroplacental dysfunction

Features

Potential consequences of pre-eclampsia

- eclampsia

    - other neurological complications include altered mental status, blindness, stroke, clonus, severe headaches or persistent visual scotomata
- fetal complications
    - intrauterine growth retardation
    - prematurity
- liver involvement (elevated transaminases)
- haemorrhage: placental abruption, intra-abdominal, intra-cerebral
- cardiac failure

Features of severe pre-eclampsia
- hypertension: typically > 160/110 mmHg and proteinuria as above
- proteinuria: dipstick ++/+++
- headache
- visual disturbance
- papilloedema
- RUQ/epigastric pain
- hyperreflexia
- platelet count < 100 * 10^6/l, abnormal liver enzymes or HELLP syndrome

Prevention

NICE divide risk factors into high and moderate risk:

High risk factors	Moderate risk factors
hypertensive disease in a previous pregnancy chronic kidney disease autoimmune disease, such as systemic lupus erythematosus or antiphospholipid syndrome type 1 or type 2 diabetes chronic hypertension	first pregnancy age 40 years or older pregnancy interval of more than 10 years body mass index (BMI) of 35 kg/m^2 or more at first visit family history of pre-eclampsia multiple pregnancy

Reducing the risk of hypertensive disorders in pregnancy

- women with the following should take aspirin 75-150mg daily from 12 weeks gestation until the birth
    - ≥ 1 high risk factors
    - ≥ 2 moderate factors

Management

Initial assessment

- NICE recommend arranging emergency secondary care assessment for any woman in whom pre-eclampsia is suspected
- women with blood pressure ≥ 160/110 mmHg are likely to be admitted and observed

Further management

- oral labetalol is now first-line following the 2010 NICE guidelines. Nifedipine (e.g. if asthmatic) and hydralazine may also be used
- delivery of the baby is the most important and definitive management step. The timing depends on the individual clinical scenario

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 20 👎 4

2019 Hypertension in pregnancy: diagnosis and management

Suggest link Report broken link

Media

Preeclampsia & eclampsia

Osmosis - YouTube 👍 1 👎 0

Pre Eclampsia - Overview

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **18.8%**

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Question 129 of 328

A 76-year-old female presents to the clinic for evaluation in the company of her daughter. She has a history of ischaemic cardiomyopathy with a recent echocardiogram showing an ejection fraction of 30%, increased filling pressures, moderate aortic stenosis, moderate mitral regurgitation, and pulmonary arterial systolic pressures of 32 mmHg. At present, she can walk approximately 100 yards before she gets significantly short of breath, and this has been the case since her last hospitalisation 4 months ago. Otherwise, she feels reasonably well and has no new complaints today. She monitors her weight regularly at home and says that these have been stable.

Her medications include aspirin, amlodipine, metoprolol, lisinopril, furosemide, gabapentin, as required paracetamol, and timolol and brinzolamide eye drops. Physical examination shows a heart rate of 63 beats per minute and blood pressure of 129/76 mmHg. Her JVP is about 4cm above the angle of Louis, her apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of her chest is clear and she has trace peripheral oedema.

What is the next best step in the management of this patient?

- ○ Discontinue amlodipine ×
- ○ Add spironolactone ×
- ○ Undertaken ECG to assess her QRS interval ×
- ○ Up-titrate the dose of furosemide ×
- ○ Go down on the dose of her metoprolol ×

Submit answer

Reference ranges ⌄

Score: **18.8%**

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Question 129 of 328 ✗

A 76-year-old female presents to the clinic for evaluation in the company of her daughter. She has a history of ischaemic cardiomyopathy with a recent echocardiogram showing an ejection fraction of 30%, increased filling pressures, moderate aortic stenosis, moderate mitral regurgitation, and pulmonary arterial systolic pressures of 32 mmHg. At present, she can walk approximately 100 yards before she gets significantly short of breath, and this has been the case since her last hospitalisation 4 months ago. Otherwise, she feels reasonably well and has no new complaints today. She monitors her weight regularly at home and says that these have been stable.

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What is the next best step in the management of this patient?

Discontinue amlodipine	8%
Add spironolactone	67%
Undertaken ECG to assess her QRS interval	19%
Up-titrate the dose of furosemide	4%
Go down on the dose of her metoprolol	2%

Patients with heart failure and an ejection fraction < 35% who are euvolaemic and are already taking an ACE-inhibitor and a beta-blocker should have an aldosterone antagonist added to their regimen as a next step

Important for me Less important

This patient has congestive heart failure related to her history of ischaemic cardiomyopathy. She seems to be doing relatively well, and her symptoms are stable. In addition, she has been monitoring her weight at home which has also been stable, and she does not have signs of volume overload on physical exam. Her heart rate and blood pressure are also in acceptable limits. Of note, her recent echocardiogram shows an ejection fraction of 30%. She, therefore, qualifies for the addition of an aldosterone antagonist to her drug regimen. Individuals with congestive heart failure who already on an ACE-inhibitor and beta-blocker, and to have an ejection fraction of less than 35% who are started on an aldosterone antagonist have been shown to have low mortality compared to those who do not have an aldosterone antagonist added to their medications.

Her blood pressure is acceptable at the moment and so she does not need to discontinue amlodipine.

Cardiac resynchronization therapy is indicated in heart failure patients with NYHA functional class III and IV patients who remain symptomatic despite stable, optimal heart failure medical therapy and who have an LVEF ≤ 35% and a prolonged QRS duration on an ECG. This patient is not yet on optimal medical therapy and should be started on an aldosterone antagonist before she can be considered for this approach.

She does not have signs of volume overload and therefore does not require changes to the dose of her furosemide today.

Her heart rate is in acceptable limits today and she does not have symptoms to suggest that the dose of her beta-blocker needs to be decreased.

👍 👎 Discuss (8) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

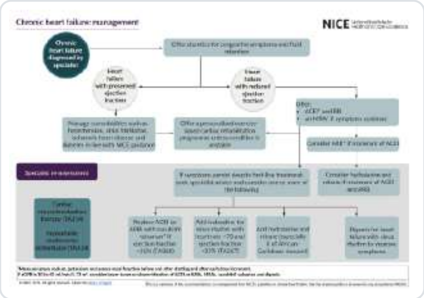
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
  - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
  - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

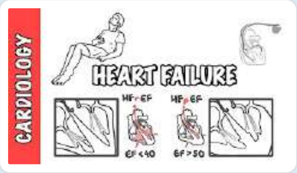
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

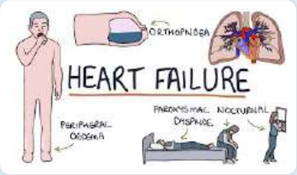
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **18.6%**

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Question 130 of 328

An 84-year-old presents to the emergency department with and acute deterioration in shortness of breath on a background of 9 months of progressively reduced exercise tolerance. Her past medical history includes hypertension, previous myocardial infarctions 7 months ago, chronic kidney disease and COPD. She has a 50 pack year smoking history giving up 2 years ago. Her exercise tolerance is 150 yards, limited by shortness of breath.

On examination, an ejection systolic murmur can be heard in the aortic area with bibasal crackles on chest auscultation. An admission chest x-ray is consistent with pulmonary oedema and intravenous diuresis is commenced. A transthoracic echocardiogram demonstrates ejection fraction of 33% with impaired left ventricular function, no vegetations, a bicuspid heavily calcified aortic valve with an area of 0.7cm2 and a peak gradient of 32mmHg. Angiography demonstrates non-flow limiting stenosis of 65% in the left anterior descending artery but no lesions requiring revascularisation. Lung function testing following successful diuresis reveals forced vital capacity at 55% of predicted and forced expiratory volume in 1 second at 48% of predicted.

The patient is keen for a definitive intervention if appropriate. What is the appropriate next action?

- ○ No action
- ○ Balloon aortic valvuloplasty
- ○ Aortic valve replacement
- ○ Transcatheter aortic valve implantation
- ○ Coronary artery bypass graft and aortic valve replacement

Submit answer

Reference ranges

Score: **18.6%**

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Question 130 of 328 ×

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The patient is keen for a definitive intervention if appropriate. What is the appropriate next action?

No action	4%
Balloon aortic valvuloplasty	15%
Aortic valve replacement	19%
🟩 Transcatheter aortic valve implantation	48%
🟥 Coronary artery bypass graft and aortic valve replacement	14%

The patient has presented with pulmonary oedema on a background of severe aortic stenosis, secondary to calcifications and a bicuspid valve. Interestingly, this is a patient who presents with low-gradient low-area aortic stenosis not uncommonly encountered in more elderly patients. In the context of poor LV function, the aortic valve gradient may be normal or only mildly raised in the presence of a severely narrowed aortic valve area.

A second consideration is the suitability of this patient for surgery. She has underlying lung disease secondary to COPD and functional impairment on pulmonary testing, renal impairment and recent myocardial infarction. She is a high-risk surgical candidate and unlikely suitable for aortic valve replacement. Inaction is not an option, the patient has presented with pulmonary oedema and underlying exercise-induced dyspnoea. Balloon aortic valvuloplasty is a palliative procedure prone to restenosis for patients unsuitable for other interventions. She does not require a CABG for her coronary artery disease, which worryingly seems inconsistent with the degree of left ventricular impairment, suggesting aortic stenosis to likely be responsible for the majority of symptoms. A transcatheter aortic valve implant (TAVI) is the appropriate management in this situation. The catheter-delivered device produces similar one-year survival as aortic valve replacement but a higher risk of stroke, TIAs and vascular complications. Compared to medical management alone, one-year survival is reduced with TAVI from 50.7% to 30.7%[1].

1. Thomas M, Schymik G, Walther T et al. One-year outcomes of cohort 1 in the Edwards SAPIEN Aortic Bioprosthesis European Outcome (SOURCE) registry: the European registry of transcatheter aortic valve implantation using the Edwards SAPIEN valve. Circulation. 2011 Jul;124(4):425-33. Epub 2011 Jul 11.

👍 👎 💬 Discuss (13) Improve

Next question >

Aortic stenosis ★

Clinical features of symptomatic disease

- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur

  - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
  - classically radiates to the carotids
  - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
  - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
  - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
  - may be used in children with no aortic valve calcification
  - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

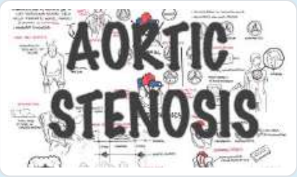
Guidelines on the management of valvular heart disease

Suggest link Report broken link

Media

Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1


AORTIC STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

AORTIC VALVE DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **18.5%**

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Question 131 of 328

A 53-year-old gentleman is reviewed in clinic due to abnormal blood tests. He has a past medical history of hypertension, type two diabetes, obesity, and depression. He started taking atorvastatin two months ago due to a routine assessment of QRISK and elevated cholesterol levels. He had a blood test as requested at three months following the start of treatment.

These shows that his alanine aminotransferase have increased from 28iU/L to 94iU/L. Other blood tests have remained within normal ranges apart from cholesterol which as improved from 5.4mmol/L to 4.9mmol/L.

How should his atorvastatin treatment be managed?

○ Stop atorvastatin and repeat LFT within 4-6 weeks ×

○ Continue atorvastatin and repeat LFT within 4-6 weeks ×

○ Change atorvastatin to simvastatin ×

○ Investigate for elevated creatinine kinase ×

○ Stop atorvastatin and arrange for an urgent abdominal ultrasound ×

Submit answer

Reference ranges

Score: **18.5%**

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2 ×

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A 53-year-old gentleman is reviewed in clinic due to abnormal blood tests. He has a past medical history of hypertension, type two diabetes, obesity, and depression. He started taking atorvastatin two months ago due to a routine assessment of QRISK and elevated cholesterol levels. He had a blood test as requested at three months following the start of treatment.

These shows that his alanine aminotransferase have increased from 28iU/L to 94iU/L. Other blood tests have remained within normal ranges apart from cholesterol which as improved from 5.4mmol/L to 4.9mmol/L.

How should his atorvastatin treatment be managed?

Stop atorvastatin and repeat LFT within 4-6 weeks	24%
Continue atorvastatin and repeat LFT within 4-6 weeks	63%
Change atorvastatin to simvastatin	8%
Investigate for elevated creatinine kinase	3%
Stop atorvastatin and arrange for an urgent abdominal ultrasound	2%

The correct answer is to continue atorvastatin and repeat LFT within 4-6 weeks. This patient has developed elevated transaminases following starting a statin, but these are within three times the upper range of normal. Please note that this is three times the maximum normal range, not three times above the patient's baseline result. Therefore it is acceptable to continue atorvastatin but monitor liver function with a repeat test in 4-6 weeks. If the level had been greater than three times the normal upper range then stopping the atorvastatin and repeating LFT would have been appropriate. There is no need to arrange for an ultrasound at the moment as there is a clear explanation, and there is no evidence of new muscle pain to justify testing for creatinine kinase. Changing statin can play a role if the statin is not tolerated, but that is not the case so far.

Source:

NICE (2014) Lipid modification: cardiovascular risk assessment and the modification of blood lipids for the primary and secondary prevention of cardiovascular disease (full guideline).National Institute for Health and Care Excellence.

👍 | 👎 | 💬 Discuss (3) | Improve

Next question >

Statins ★

Statins inhibit the action of HMG-CoA reductase, the rate-limiting enzyme in hepatic cholesterol synthesis.

Adverse effects
- myopathy: includes myalgia, myositis, rhabdomyolysis and asymptomatic raised creatine kinase. Risks factors for myopathy include advanced age, female sex, low body mass index and presence of multisystem disease such as diabetes mellitus. Myopathy is more common in lipophilic statins (simvastatin, atorvastatin) than relatively hydrophilic statins (rosuvastatin, pravastatin, fluvastatin)
- liver impairment: the 2014 NICE guidelines recommend checking LFTs at baseline, 3 months and 12 months. Treatment should be discontinued if serum transaminase concentrations rise to and persist at 3 times the upper limit of the reference range
- there is some evidence that statins may increase the risk of intracerebral haemorrhage in patients who've previously had a stroke. This effect is not seen in primary prevention. For this reason the Royal College of Physicians recommend avoiding statins in patients with a history of intracerebral haemorrhage

Contraindications
- macrolides (e.g. erythromycin, clarithromycin) are an important interaction. Statins should be stopped until patients complete the course
- pregnancy

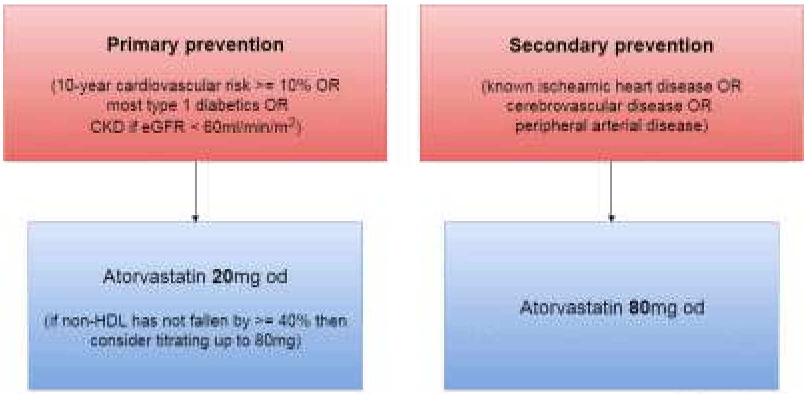
Who should receive a statin?

- all people with established cardiovascular disease (stroke, TIA, ischaemic heart disease, peripheral arterial disease)
- following the 2014 update, NICE recommend anyone with a 10-year cardiovascular risk >= 10%
- patients with type 2 diabetes mellitus should now be assessed using QRISK2 like other patients are, to determine whether they should be started on statins
- patients with type 1 diabetes mellitus who were diagnosed more than 10 years ago OR are aged over 40 OR have established nephropathy

Statins should be taken at night as this is when the majority of cholesterol synthesis takes place. This is especially true for simvastatin which has a shorter half-life than other statins.

NICE currently recommends the following for the prevention of cardiovascular disease::

- atorvastatin 20mg for primary prevention
    - increase the dose if non-HDL has not reduced for >= 40%
- atorvastatin 80mg for secondary prevention


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od

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Graphic showing choice of statin.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 15 👎 21

2016 Stroke guidelines

NICE 👍 20 👎 16

2014 Lipid modification guidelines

Clinical Knowledge Summaries 👍 18 👎 14

Lipids management

NICE 👍 18 👎 12

2022 Type 2 diabetes guidelines

Suggest link Report broken link

Media

HMG-CoA Reductase Inhibitors
STATINS

Drugs for hyperlipidaemia

Speed Pharmacology - YouTube 👍 3 👎 0

Report broken media

Score: **18.3%**

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A 28 year-old man with a background of asthma presents to the emergency department with palpitations.

He reports a 2 day history of coryzal symptoms and wheeze. He has been using his salbutamol inhaler several times per day due to these symptoms.

On examination the patient appears alert and comfortable but complains of a fluttering sensation in his chest. The pulse rate is 180bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on room air.

There is equal bilateral air entry with mild polyphonic wheeze throughout on chest auscultation.

A 12-lead ECG reveals a narrow-complex regular tachycardia at 180bpm with pseudo r' waves after each QRS complex in lead V1.

What is the most appropriate initial management?

○ Adenosine 6mg IV
○ Metoprolol 5mg IV
○ Synchronised DC shock
○ Vagal manoeuvres
○ Amiodarone 300mg IV

Submit answer

Reference ranges

← Question 132 of 328 ✗ ⚐ →

A 28 year-old man with a background of asthma presents to the emergency department with palpitations.

He reports a 2 day history of coryzal symptoms and wheeze. He has been using his salbutamol inhaler several times per day due to these symptoms.

On examination the patient appears alert and comfortable but complains of a fluttering sensation in his chest. The pulse rate is 180bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on room air.

There is equal bilateral air entry with mild polyphonic wheeze throughout on chest auscultation.

A 12-lead ECG reveals a narrow-complex regular tachycardia at 180bpm with pseudo r' waves after each QRS complex in lead V1.

What is the most appropriate initial management?

Adenosine 6mg IV	8%
Metoprolol 5mg IV	2%
Synchronised DC shock	1%
Vagal manoeuvres	83%
Amiodarone 300mg IV	6%

> The first-line management of SVT is vagal manoeuvres: e.g. Valsalva manoeuvre or carotid sinus massage
>
> Important for me Less important

This scenario describes a supraventricular tachycardia, most likely to be atrioventricular nodal reentrant tachycardia (AVNRT). AV nodal reentrant tachycardia is the most common regular supraventricular tachycardia.

Often a retrograde p-wave is visible in continuity with the QRS complex, appearing as a 'pseudo r'' wave in lead V1 or a 'pseudo s'' wave in the inferior leads.

According to the Resuscitation council guidelines, a regular narrow-complex tachycardia in the absence of adverse features (shock, syncope, myocardial ischaemia or heart failure) should be treated initial with vagal manoeuvres. Adenosine and metoprolol may worsen bronchospasm so would not be appropriate in this setting. IV Verapamil or synchronised DC cardioversion could be tried if vagal manoeuvres are unsuccessful.

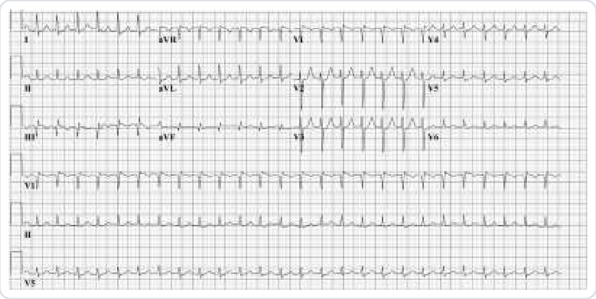
Source: https://www.resus.org.uk/resuscitation-guidelines/peri-arrest-arrhythmias/#tachycardia

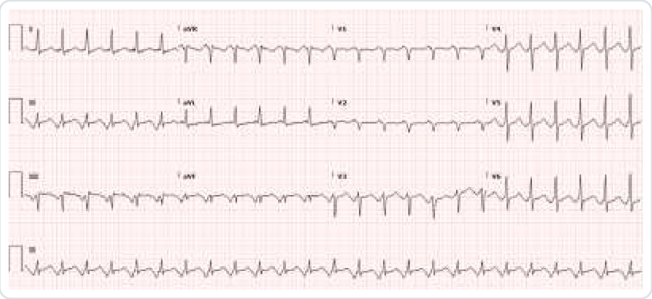
👍 👎 Discuss (4) Improve

Next question >

Supraventricular tachycardia

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
    - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
    - carotid sinus massage
- intravenous adenosine
    - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
    - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

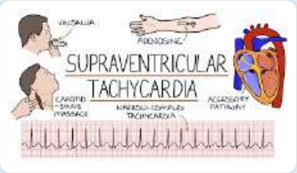
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

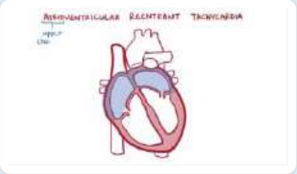
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR
TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

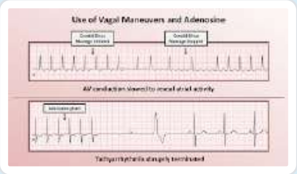
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0


Use of Vagal Maneuvers and Adenosine

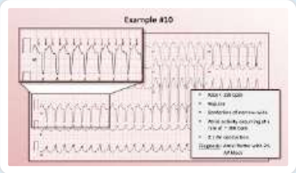
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0

ATRIOVENTRICULAR NODAL RE-ENTRANT TACHYCARDIA (AVNRT)
AVRT & AVNRT
Khan Academy

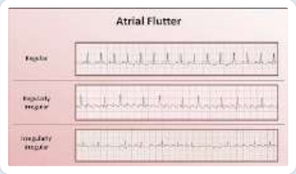
[Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)]

Khan Academy - YouTube 👍 2 👎 0



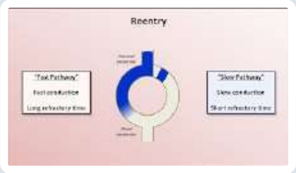
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0


Atrial Flutter
Regular
Regularly Irregular
Irregularly Irregular

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **18.2%**

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Question 133 of 328

A 45-year-old male is referred to the rapid access chest pain clinic with symptoms typical of angina. He is a non-smoker and has no past medical history. The pain is located in the centre of his chest and made worse by exertion. According to the NICE guidelines, which is the most appropriate investigation?

○ Treadmill ECG ×

○ Trans-thoracic echocardiogram ×

○ CT-coronary angiography ×

○ Nuclear perfusion stress test ×

○ Diagnostic coronary angiography ×

Submit answer

Reference ranges

Score: **18.2%**

1	✗
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A 45-year-old male is referred to the rapid access chest pain clinic with symptoms typical of angina. He is a non-smoker and has no past medical history. The pain is located in the centre of his chest and made worse by exertion. According to the NICE guidelines, which is the most appropriate investigation?

Treadmill ECG	17%
Trans-thoracic echocardiogram	3%
CT-coronary angiography	67%
Nuclear perfusion stress test	5%
Diagnostic coronary angiography	9%

The correct answer is **CT-coronary angiography**. According to the NICE guidelines (CG95), a CT coronary angiogram should be offered as the first-line investigation for patients with typical or atypical angina symptoms and who are in the intermediate risk group (10-29% 10-year risk of developing cardiovascular disease). This patient fits into this category due to his age, sex, and lack of other risk factors. The CT coronary angiogram will help visualise the coronary arteries and identify any significant stenosis.

Treadmill ECG was once a common initial test for angina but it has now been superseded by more accurate tests such as CT coronary angiography. Treadmill ECG can have false positives and negatives, hence it is not recommended as a first-line investigation by NICE.

A **Trans-thoracic echocardiogram** is useful in assessing cardiac function but it does not directly visualise the coronary arteries so it would not be appropriate in this case. It may be used in conjunction with other tests if there are signs of heart failure or valvular heart disease.

Nuclear perfusion stress test involves injecting a radioactive tracer and then imaging the heart under stress and at rest. While this test can identify areas of myocardium that are under-perfused due to significant stenosis, it exposes patients to radiation, is more expensive than CT angiography and does not directly image the coronaries. Hence, it is usually reserved for cases where other investigations are contraindicated or inconclusive.

Finally, **Diagnostic coronary angiography**, while considered the gold standard for diagnosing coronary artery disease, is invasive and carries risks such as bleeding, stroke or kidney damage from contrast dye. As per NICE guidelines, it should only be used when non-invasive tests are inconclusive or contraindicated or when immediate revascularisation is being considered.

👍 👎 Discuss (5) Improve

Next question >

Chest pain: assessment of patients with suspected cardiac chest pain

NICE updated it's guidelines in 2016 on the 'Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin'.

Below is a brief summary of the key points. Please see the link for more details.

Patients presenting with acute chest pain

Immediate management of suspected acute coronary syndrome (ACS)
- glyceryl trinitrate
- aspirin 300mg. NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital
- do not routinely give oxygen, only give if sats < 94%*
- perform an ECG as soon as possible but do not delay transfer to hospital. A normal ECG does not exclude ACS

Referral

- current chest pain or chest pain in the last 12 hours with an abnormal ECG: emergency admission
- chest pain 12-72 hours ago: refer to hospital the same-day for assessment
- chest pain > 72 hours ago: perform full assessment with ECG and troponin measurement before deciding upon further action

*NICE suggest the following in terms of oxygen therapy:
- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is available.

Patients presenting with stable chest pain

NICE define anginal pain as the following:
- 1. constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arms
- 2. precipitated by physical exertion
- 3. relieved by rest or GTN in about 5 minutes
- patients with all 3 features have typical angina
- patients with 2 of the above features have atypical angina
- patients with 1 or none of the above features have non-anginal chest pain

For patients in whom stable angina cannot be excluded by clinical assessment alone NICE recommend the following (e.g. symptoms consistent with typical/atypical angina OR ECG changes):
- 1st line: CT coronary angiography
- 2nd line: non-invasive functional imaging (looking for reversible myocardial ischaemia)
- 3rd line: invasive coronary angiography

Examples of non-invasive functional imaging:
- myocardial perfusion scintigraphy with single photon emission computed tomography (MPS with SPECT) or

- stress echocardiography or
- first-pass contrast-enhanced magnetic resonance (MR) perfusion or
- MR imaging for stress-induced wall motion abnormalities

+ Q 123

Next question >

B I A

Textbooks

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Links

European Society of Cardiology 👍 14 👎 7

2013 ESC guidelines on the management of stable coronary artery disease

NICE 👍 11 👎 3

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Suggest link Report broken link

Score: **18%**

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A 68-year-old male is admitted with confusion and an ataxic gait. His past medical history includes heart failure and atrial fibrillation. His drug history includes dabigatran, bisoprolol and ramipril.

Investigation results are as follows:

Hb	110 g/l	Na^+	139 mmol/l
Platelets	140 * 10^9/l	K^+	3.8 mmol/l
WBC	10.2 * 10^9/l	Urea	6.5 mmol/l
Neuts	8.2 * 10^9/l	Creatinine	42 µmol/l
Lymphs	1.4 * 10^9/l	CRP	12 mg/l
PT ratio	1.6 * 10^9/l	aPTT	50 seconds

CT head	Large acute right-sided subdural haematoma with ventricular effacement and midline shift

How will you manage this patient?

- ○ Platelet transfusion
- ○ Fresh frozen plasma
- ○ Red cell transfusion
- ○ Tranexamic acid
- ○ Idarucizumab

Submit answer

Reference ranges

A 68-year-old male is admitted with confusion and an ataxic gait. His past medical history includes heart failure and atrial fibrillation. His drug history includes dabigatran, bisoprolol and ramipril.

Investigation results are as follows:

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Lymphs	1.4 * 10^9/l	CRP	12 mg/l
PT ratio	1.6 * 10^9/l	aPTT	50 seconds

CT head	Large acute right-sided subdural haematoma with ventricular effacement and midline shift

How will you manage this patient?

Platelet transfusion	1%
Fresh frozen plasma	11%
Red cell transfusion	0%
Tranexamic acid	2%
Idarucizumab	85%

Bleeding on dabigatran? Can use idarucizumab to reverse

Important for me Less important

Dabigatran is an oral anticoagulant acting by direct inhibition of thrombin. In active bleeding, or life-threatening haemorrhage, idarucizumab should be used. This is a specific antidote which reverses the effect of dabigatran.

The PT ratio and INR is not a reliable marker following dabigatran ingestion, and should not be used to monitor the anticoagulant effects. The aPTT and, if available, the thrombin time (TT) should be used to measure the anticoagulant effect of dabigatran,

If idarucizumab is unavailable, and particularly if there is evidence of acquired coagulopathy, consider administration of red cell concentrate, fresh frozen plasma, and/or platelet transfusion.

👍 | 👎 | Discuss (4) | Improve

Next question >

Dabigatran ★

Dabigatran is an oral anticoagulant that works by being a direct thrombin inhibitor. It is one of the drugs developed over the past 20 years as an alternative to warfarin, with the advantage that it does not require regular monitoring.

What is dabigatran used for?

Dabigatran is currently used for two main indications.

Firstly it is an option in the prophylaxis of venous thromboembolism following hip or knee replacement surgery.

Secondly, it is also licensed in the UK for prevention of stroke in patients with non-valvular atrial fibrillation who have one or more of the following risk factors present:

- previous stroke, transient ischaemic attack or systemic embolism
- left ventricular ejection fraction below 40%
- symptomatic heart failure of New York Heart Association (NYHA) class 2 or above
- age 75 years or older
- age 65 years or older with one of the following: diabetes mellitus, coronary artery disease or hypertension

What are the known side-effects of dabigatran?

Unsurprisingly haemorrhage is the major adverse effect.

Doses should be reduced in chronic kidney disease and dabigatran should not be prescribed if the creatinine clearance is < 30 ml/min.

Reversing the effects

Idarucizumab cab be used for rapid reversal of the anticoagulant effects of dabigatran.

Drug Safety Update 2013

The RE-ALIGN study showed significantly higher bleeding and thrombotic events in patients with recent mechanical heart valve replacement using dabigatran compared with warfarin.

Previously there had been no guidance to support the use of dabigatran in patients with prosthetic heart valves but the advice has now changed to dabigatran being contraindicated in such patients.

+ Q 123

Next question >

Textbooks

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Links

Medicines and Healthcare products Regulatory Agency 👍 8 👎 6

Drug Safety Update - March 2013

Suggest link

Report broken link

Score: **18.7%**

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An 18-year-old male presents with shortness of breath. He states that it has gradually worsened over the past year. He denies cough, wheeze or chest pain. He has no past medical history of note. On examination you note a loud second heart sound. An ECG shows right bundle branch block (RBBB) with left axis deviation (LAD).

What is the most likely diagnosis?

- ○ Coronary sinus atrial septal defect ×
- ○ Ventricular septal defect ×
- ○ Idiopathic pulmonary hypertension ×
- ○ Chronic pulmonary emboli ×
- ○ Ostium primum atrial septal defect ×

Submit answer

Reference ranges

Score: **18.7%**

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Question 135 of 328 ✓

An 18-year-old male presents with shortness of breath. He states that it has gradually worsened over the past year. He denies cough, wheeze or chest pain. He has no past medical history of note. On examination you note a loud second heart sound. An ECG shows right bundle branch block (RBBB) with left axis deviation (LAD).

What is the most likely diagnosis?

Coronary sinus atrial septal defect	8%
Ventricular septal defect	13%
Idiopathic pulmonary hypertension	24%
Chronic pulmonary emboli	3%
Ostium primum atrial septal defect	53%

Both primum and secondum atrial septal defects result in RBBB. The axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secondums (usually have RAD).

Important for me Less important

The patient has presented with features of pulmonary hypertension, namely the progressive shortness of breath and loud second heart sound. The ECG is very suggestive of an atrial septal defect (ASD). Atrial septal defects result in RBBB. The axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secondums (usually have RAD). The ECG features in this case are therefore more suggestive of an ostium primum.

Furthermore, although ostium secondums are more common overall, they do not usually present as early as this. In contrast, ostium primum defects are usually much lower down in the septum and often involve the atrioventricular valves which accelerates the pathogenesis resulting in presentation at a younger age.

👍 👎 Discuss (10) Improve

Next question ›

Atrial septal defects ★

Atrial septal defects (ASDs) are the most likely congenital heart defect to be found in adulthood. They carry a significant mortality, with 50% of patients being dead at 50 years. Two types of ASDs are recognised, ostium secundum and ostium primum. Ostium secundum are the most common

Features
- ejection systolic murmur, fixed splitting of S2
- embolism may pass from venous system to left side of heart causing a stroke

Ostium secundum (70% of ASDs)
- associated with Holt-Oram syndrome (tri-phalangeal thumbs)
- ECG: RBBB with RAD

Ostium primum
- present earlier than ostium secundum defects
- associated with abnormal AV valves
- ECG: RBBB with LAD, prolonged PR interval

AI + Q 123

Next question ›

B *I*

Textbooks

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Media

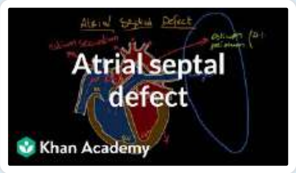
RA
Left atrium
ASD
RV
LV
VSD
Pulmonary artery
aorta
PDA

How to diagnose acyanotic heart diseases (Left to Right shunt) via Cardiac Catheterization.

Medicosis Perfectionalis - YouTube 👍 2 👎 0

Atrial septal defect murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1


Atrial Septal Defect
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Khan Academy

Atrial septal defects

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ATRIAL SEPTAL DEFECT
Osmosis.org

Atrial septal defect

Osmosis - YouTube 👍 6 👎 4

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Score: **19.3%**

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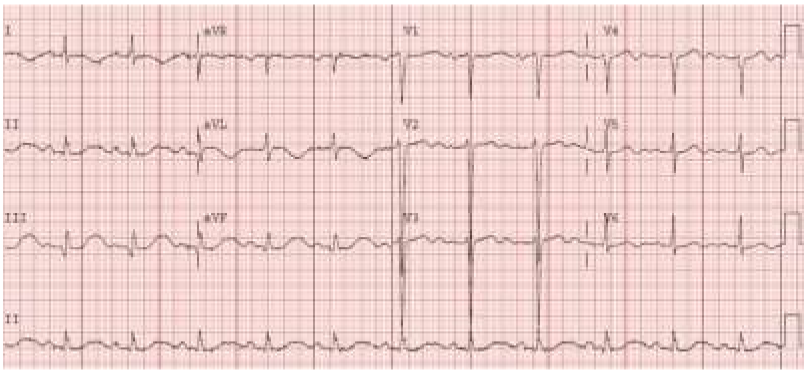
A 23-year-old man with known ulcerative colitis presents for the fourth time in 6 months with a severe flare. On examination, he has a distended and generally tender abdomen. He was treated with intravenous hydrocortisone, standard 0.9% saline intravenous fluids and thromboprophylaxis. Although an abdominal film demonstrated dilated large bowel loops, no operation was deemed necessary after surgical review. The same treatment was continued for 3 days. On day 3 of his admission, he reports chest pain with palpations. A 12 lead ECG was taken:



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What is the most likely underlying diagnosis?

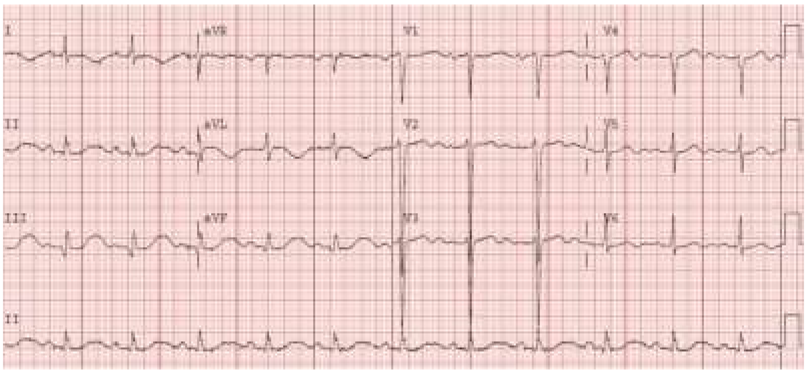
- ○ Hyperkalaemia
- ○ Hypokalaemia
- ○ Hypocalcaemia
- ○ Intravascular depletion → sinus tachycardia
- ○ Atrial fibrillation

Submit answer

Reference ranges

Question 136 of 328

A 23-year-old man with known ulcerative colitis presents for the fourth time in 6 months with a severe flare. On examination, he has a distended and generally tender abdomen. He was treated with intravenous hydrocortisone, standard 0.9% saline intravenous fluids and thromboprophylaxis. Although an abdominal film demonstrated dilated large bowel loops, no operation was deemed necessary after surgical review. The same treatment was continued for 3 days. On day 3 of his admission, he reports chest pain with palpations. A 12 lead ECG was taken:



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What is the most likely underlying diagnosis?

Hyperkalaemia	5%
Hypokalaemia	80%
Hypocalcaemia	12%
Intravascular depletion → sinus tachycardia	2%
Atrial fibrillation	1%

Hypokalaemia is a common consequence following extensive GI losses, in addition to bicarbonate and chloride losses. In this case, lack of potassium replacement resulted in tachyarrhythmia with U waves (see extra complex after the T wave). The patient may well be intravenously dry as well but while this may result in tachycardia, it does not explain U waves.

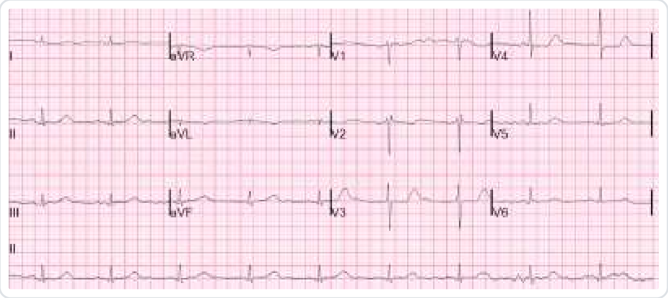
Discuss (6) | Improve

Next question >

ECG: hypokalaemia ★

ECG features of hypokalaemia

- U waves
- small or absent T waves (occasionally inversion)
- prolong PR interval
- ST depression
- long QT



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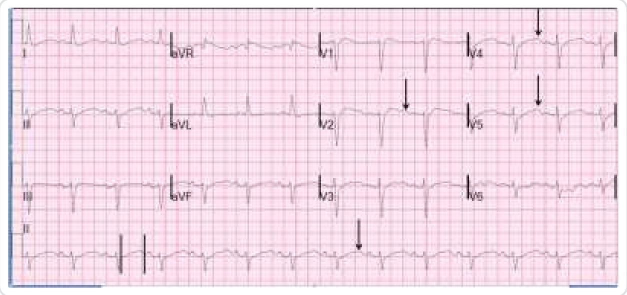

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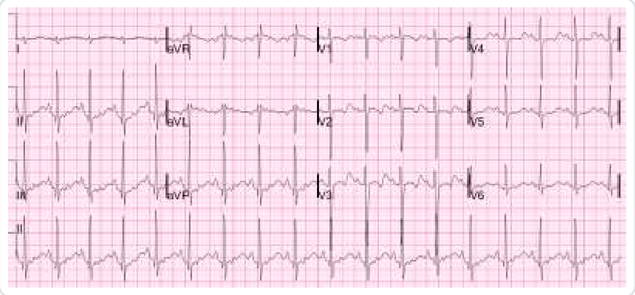

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In Hypokalaemia, U have no Pot and no T, but a long PR and a long QT

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 48 👎 6

ECG: Hypokalaemia changes

Suggest link

Report broken link

Score: **19.1%**

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Question 137 of 328

A 78-year-old woman presents to the emergency department with complaints of palpitations. Her episodes of palpitations occur roughly once a month and are not associated with any chest pain or shortness of breath. She has a past medical history of ischaemic heart disease, type 2 diabetes, depression, hypertension and migraines. She takes aspirin, metformin, simvastatin and uses paracetamol when she needs them for headaches. She lives alone with a twice daily package of care.

An ECG demonstrates an irregularly irregular rhythm with no p-waves. She is offered bisoprolol to control her symptoms but does not want to take it regularly. She asks if she could take the bisoprolol only when she has palpitations as they occur so rarely. What would make a 'pill-in-the-pocket'□ strategy inappropriate for her?

- ○ Hypertension ×
- ○ History of ischaemic heart disease ×
- ○ Age ×
- ○ Living alone ×
- ○ Palpitations too frequent ×

Submit answer

Reference ranges

Score: **19.1%**

1 ×
2 ×

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Back to top

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Hypertension	3%
✓ History of ischaemic heart disease	50%
Age	10%
Living alone	27%
Palpitations too frequent	10%

The correct answer is a history of ischaemic heart disease. This patient has paroxysmal AF. NICE recommends that when people have infrequent paroxysms and few symptoms a 'pill-in-the-pocket' strategy can be offered. This means giving rate-control medications for as needed use, rather than regular use. It can be considered in those with paroxysmal AF who:

- Have no history of left ventricular dysfunction, valvular or ischaemic heart disease
- Have infrequent episodes
- Systolic blood pressure >100mmHg and heart rate >70bpm
- Are able to understand when to take the tablet

This patient could not be offered this strategy as she has a history of ischaemic heart disease.

Source:
'Atrial Fibrillation: Management.' The National Institute for Health and Care Excellence, June 2014.

Discuss (12) Improve

Next question >

Atrial fibrillation: a very basic introduction ★

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. It is very common, being present in around 5% of patients over aged 70-75 years and 10% of patients aged 80-85 years. Whilst uncontrolled atrial fibrillation can result in symptomatic palpitations and inefficient cardiac function probably the most important aspect of managing patients with AF is reducing the increased risk of stroke which is present in these patients.

Types of atrial fibrillation

AF may by classified as either first detected episode, paroxysmal, persistent or permanent.

- first detected episode (irrespective of whether it is symptomatic or self-terminating)
- recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term **paroxysmal AF** is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term **persistent AF** is used. Such episodes usually last greater than 7 days
- in **permanent AF** there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rated control and anticoagulation if appropriate

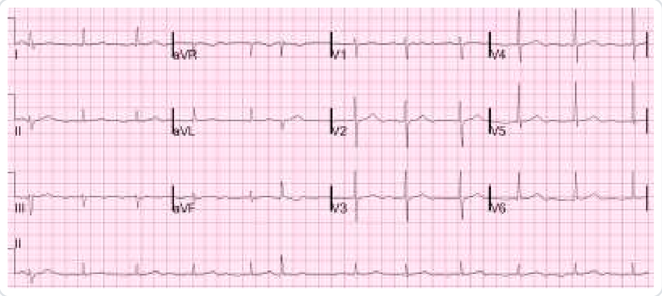
Symptoms and signs

Symptoms
- palpitations
- dyspnoea
- chest pain

Signs
- an irregularly irregular pulse

Investigations

An ECG is essential to make the diagnosis as other conditions can give an irregular pulse, such as ventricular ectopics or sinus arrhythmia.



Management

There are two key parts of managing patients with AF:
- 1. Rate/rhythm control
- 2. Reducing stroke risk

Rate vs. rhythm control

There are two main strategies employed in dealing with the arrhythmia element of atrial fibrillation:
- **rate control**: accept that the pulse will be irregular, but slow the rate down to avoid negative effects on cardiac function
- **rhythm control**: try to get the patient back into, and maintain, normal sinus rhythm. This is termed cardioversion. Drugs (pharmacological cardioversion) and synchronised DC electrical shocks (electrical cardioversion) may be used for this purpose

For many years the predominant approach was to try and maintain a patient in sinus rhythm. This approach changed in the early 2000's and now the majority of patients are managed with a rate control strategy. NICE advocate using a rate control strategy except in a number of specific situations such as coexistent heart failure, first onset AF or where there is an obvious reversible cause.

Rate control

A **beta-blocker** or a **rate-limiting calcium channel blocker** (e.g. diltiazem) is used first-line to control the rate in AF.

If one drug does not control the rate adequately NICE recommend combination therapy with any 2 of the following:
- a betablocker
- diltiazem
- digoxin

Rhythm control

As mentioned above there are a subgroup of patients for whom a rhythm control strategy should be tried first. Other patients may have had a rate control strategy initially but switch to rhythm control if symptoms/heart rate fails to settle.

When considering cardioversion it is very important to remember that the moment a patient switches from AF to sinus rhythm presents the highest risk for embolism leading to stroke. Imagine the thrombus formed in the fibrillating atrium suddenly being pushed out when sinus rhythm is restored. For this reason patients must either have had a short duration of symptoms (less than 48 hours) or be anticoagulated for a period of time prior to attempting cardioversion.

Reducing stroke risk

Some patients with AF are at a very low risk of stroke whilst others are at a very significant risk. Clinicians use risk stratifying tools such as the **CHA_2DS_2-VASc** score to determine the most appropriate anticoagulation strategy.

	Risk factor	Points
C	Congestive heart failure	1
H	Hypertension (or treated hypertension)	1
A_2	Age >= 75 years	2
	Age 65-74 years	1
D	Diabetes	1
S_2	Prior Stroke, TIA or thromboembolism	2
V	Vascular disease (including ischaemic heart disease and peripheral arterial disease)	1
S	Sex (female)	1

The table below shows a suggested anticoagulation strategy based on the score:

Score	Anticoagulation
0	No treatment
1	Males: Consider anticoagulation Females: No treatment (this is because their score of 1 is only reached due to their gender)
2 or more	Offer anticoagulation

NICE recommend that we offer patients a choice of anticoagulation, including warfarin and the novel oral anticoagulants (NOACs).

Next question

Textbooks

High-yield textbook

Extended textbook

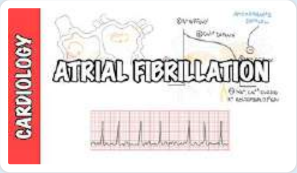
Links

NICE 👍 16 👎 7

2021 Atrial fibrillation guidelines

Suggest link Report broken link

Media

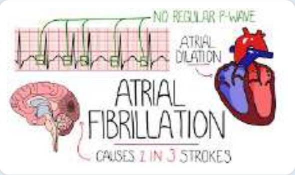

CARDIOLOGY
ATRIAL FIBRILLATION

Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 4 👎 0

Atrial fibrillation

Osmosis - YouTube 👍 1 👎 0


NO REGULAR P-WAVE
ATRIAL DILATION
ATRIAL FIBRILLATION
CAUSES 1 IN 3 STROKES

Atrial Fibrillation Made Easy

Rhesus Medicine - YouTube

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Score: **19.7%**

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137 ✓

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Back to top

A 73-year-old gentleman was shown to have left ventricular dysfunction and hypertrophy on an echocardiogram post acute myocardial infarction (MI). Prior to discharge, he was started on ramipril 2.5mg, bisoprolol 2.5mg, atorvastatin 10mg and aspirin 75mg. His medical history included osteoporosis for which he took Calcichew D3 forte and alendronic acid. He did not smoke or drink alcohol. On admission his kidney function showed the following:

Sodium	136 mmol/L
Potassium	3.7 mmol/L
Urea	7.0 mmol/L
Creatinine	120 micromol/L
eGFR	64 ml/min/1.73m²

His GP checked his bloods again ten days after the initiation of his new medication to titrate the dose of ramipril as requested on the discharge letter from the cardiology team. The results are below:

Sodium	134 mmol/L
Potassium	4.2 mmol/L
Urea	8.0 mmol/L
Creatinine	156 micromol/L
eGFR	50 ml/min/1.73m²

How would you manage this gentleman's deteriorating renal function?

- ○ Continue the ramipril and repeat the bloods in one week ×
- ○ Decrease the dose of ramipril to 1.25mg daily ×
- ○ Stop the ramipril ×
- ○ Switch the ramipril to losartan ×

○ Request a renal ultrasound scan ✕

Submit answer

Reference ranges ∨

Score: **19.7%**

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Question 138 of 328 ✗

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How would you manage this gentleman's deteriorating renal function?

Continue the ramipril and repeat the bloods in one week	72%
Decrease the dose of ramipril to 1.25mg daily	8%
Stop the ramipril	11%
Switch the ramipril to losartan	4%

Request a renal ultrasound scan 5%

NICE guidelines are useful for everyday practice like managing chronic kidney disease (CKD). Knowing the major points about recent guidelines will also help you in your revision for exams. Guideline 73 is about the diagnosis and management of CKD, the link is below.

It is useful for staging CKD and guiding typical frequency of blood monitoring. In our gentleman's case, his renal function deteriorates due to starting ramipril. NICE guidance says it is acceptable to have a 25% reduction in eGFR OR a 30% rise in creatinine after starting an ACE inhibitor or angiotensin receptor blocker.

👍 👎 Discuss (8) Improve

Next question >

ACE inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in the secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibits the conversion angiotensin I to angiotensin II
    - → decrease in angiotensin II levels → to vasodilation and reduced blood pressure
    - → decrease in angiotensin II levels → reduced stimulation for aldosterone release → decrease in sodium and water retention by the kidneys
- renoprotective mechanism
    - angiotensin II constricts the efferent glomerular arterioles
    - ACE inhibitors therefore lead to dilation of the efferent arterioles → reduced glomerular capillary pressure → decreased mechanical stress on the delicate filtration barriers of the glomeruli
    - this is particularly important in diabetic nephropathy

- ACE inhibitors are activated by phase 1 metabolism in the liver

Side-effects:

- cough
  - occurs in around 15% of patients and may occur up to a year after starting treatment
  - thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications

- pregnancy and breastfeeding - avoid
- renovascular disease - may result in renal impairment
- aortic stenosis - may result in hypotension
- hereditary of idiopathic angioedema
- specialist advice should be sought before starting ACE inhibitors in patients with a potassium >= 5.0 mmol/L

Interactions

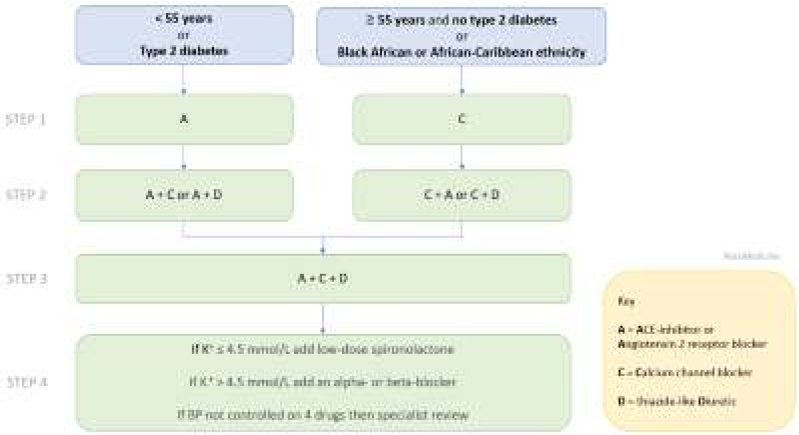
- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day)
  - significantly increases the risk of hypotension

Monitoring

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
  - a rise in the creatinine and potassium may be expected after starting ACE inhibitors
  - acceptable changes are an increase in serum creatinine, up to 30% from baseline and an increase in potassium up to 5.5 mmol/l.
  - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis

Examples of common ACE inhibitors

- ramipril
- enalapril
- lisinopril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or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
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STEP 1
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C + A or C + D
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If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 53 34

2014 Chronic kidney disease guidelines

BNF 50 25

ACE inhibitors

Suggest link Report broken link

Score: **19.6%**

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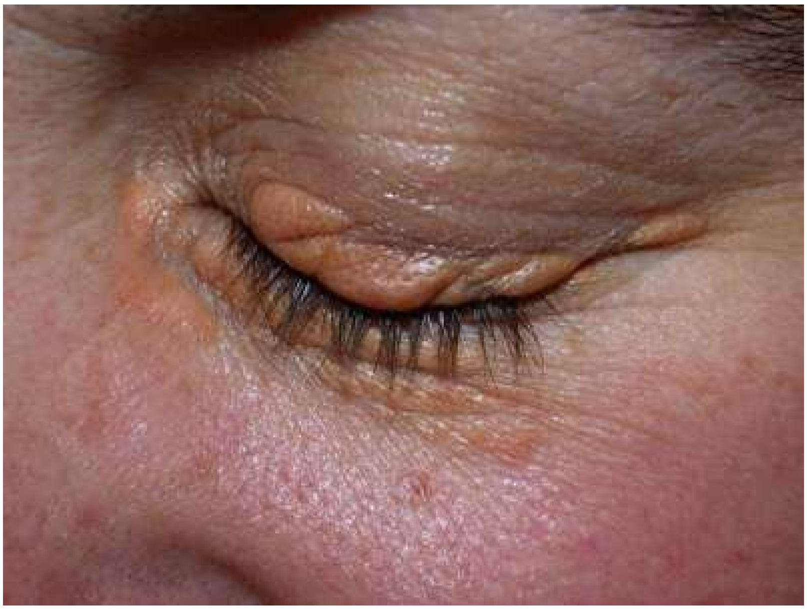
A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:



© Image used on license from DermNet NZ

Given the likely diagnosis, which of the following would be an appropriate management option?

○ Topical emollient

○ Topical hydrocortisone

○ Topical ketoconazole

○ Topical trichloroacetic acid

○ Urgent referral to secondary care

Submit answer

A 38-year-old woman is referred to the cardiology outpatient department with exertional shortness of breath. She has no past medical history.

On examination, the jugular venous pulse is elevated and there is a loud P2 component of the second heart sound.

A transthoracic echocardiogram reveals an elevated mean pulmonary artery pressure (mPAP) of 54 mmHg, which is confirmed with a right heart catheterization (RHC). Acute vasodilator testing at the time of RHC demonstrates a decrease in mPAP to 28 mmHg.

A set of comprehensive investigations does not reveal an identifiable cause of the abnormalities.

Given her diagnosis and subsequent investigations, what class of medications are appropriate to treat her illness?

- ○ Calcium channel blockers ×
- ○ Endothelin receptor antagonists ×
- ○ Phosphodiesterase inhibitors ×
- ○ Prostacyclin analogues ×
- ○ Vitamin K antagonists ×

Submit answer

Reference ranges

Score: **19.6%**

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- ○ Prostacyclin analogues ×
- ○ Vitamin K antagonists ×

Submit answer

Reference ranges

Score: **19.6%**

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Question 140 of 328 ✓

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A set of comprehensive investigations does not reveal an identifiable cause of the abnormalities.

Given her diagnosis and subsequent investigations, what class of medications are appropriate to treat her illness?

Calcium channel blockers	76%
Endothelin receptor antagonists	12%
Phosphodiesterase inhibitors	8%
Prostacyclin analogues	4%
Vitamin K antagonists	0%

Pulmonary arterial hypertension patients with positive response to vasodilator testing should be treated with calcium channel blockers

Important for me Less important

Calcium channel blockers is the correct answer. The patient has pulmonary hypertension. The gold standard investigation for the investigation of pulmonary hypertension is right heart catheterization. A mean pulmonary artery pressure >= 25 mmHg at rest defines pulmonary hypertension. Patients with pHTN should undergo vasodilator testing. A positive response to vasodilator testing as evidenced by a decline in mPAP suggests the patient may benefit from a calcium channel blocker. An inability to find a secondary cause of her disease suggests she has the idiopathic form of the illness.

Endothelin receptor antagonists is incorrect. An example of this type of medication is bosentan. This class of medication is used in those patients with negative acute vasodilator testing.

Phosphodiesterase inhibitors is not the right answer. An example of this class of medication is sildenafil. It can be used orally. This class of medication is used in those patients with negative acute vasodilator testing. Examples of vasodilators include IV epoprostenol, IV adenosine, or inhaled nitric oxide.

Prostacyclin analogues is incorrect. An example of this class of medication is iloprost. This is given as an infusion. It is used in those patients with negative acute vasodilator testing.

Vitamin K antagonists is not the right answer. An example of this class of medication is warfarin. Chronic thromboembolic disease is a cause of pulmonary hypertension but we are told a comprehensive set of investigations did not reveal a cause of pulmonary hypertension and therefore anticoagulation is not indicated.

👍 👎 Discuss (1) Improve

Next question >

Pulmonary arterial hypertension ★

Pulmonary arterial hypertension (PAH) may be defined as a resting mean pulmonary artery pressure of >= 20 mmHg. Endothelin is thought to play a key role in the pathogenesis of PAH. It is more common in females and typically presents between the ages of 30-50 years. Pulmonary hypertension may of course develop secondary to chronic lung diseases such as COPD - PAH is diagnosed in the absence of this although certain factors increase the risk, including HIV, cocaine and anorexigens (e.g. fenfluramine). Around 10% of cases are inherited in an autosomal dominant fashion.

Features

- progressive exertional dyspnoea is the classical presentation
- other possible features include exertional syncope, exertional chest pain and peripheral oedema
- cyanosis
- clinical signs:
  - right ventricular heave: indicating right ventricular hypertrophy or dilatation
  - loud P2: early in the disease reflects increased pulmonary artery pressure and may be accompanied by a palpable P2 in severe cases. With advanced PAH there may be right ventricular failure leading to a soft S2
  - raised JVP with prominent 'a' waves: reflects increased resistance to right atrial emptying due to elevated right ventricular end-diastolic pressure
  - tricuspid regurgitation

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that **acute vasodilator testing** is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide.

If there is a positive response to acute vasodilator testing (a minority of patients)

- oral calcium channel blockers

If there is a negative response to acute vasodilator testing (the vast majority of patients)

- prostacyclin analogues: treprostinil, iloprost
- endothelin receptor antagonists
  - non-selective: bosentan
  - selective antagonist of endothelin receptor A: ambrisentan
- phosphodiesterase inhibitors: sildenafil

Patients with progressive symptoms should be considered for a heart-lung transplant.

＋ Q 123

Next question >

B *I* **A** T↕

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 13

2015 Guidelines for the diagnosis and treatment of pulmonary hypertension

Medcomic 👍 14 👎 8

Pulmonary hypertension

Respiratory Medicine journal 👍 13 👎 7

Pulmonary vasodilator testing and use of calcium channel blockers in pulmonary arterial hypertension

Suggest link Report broken link

Media

Pulmonary hypertension

Osmosis - YouTube 👍 1 👎 4

Report broken media

Score: **20.1%**

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Question 141 of 328

A 68-year-old Afro-Caribbean gentleman is short of breath on dressing, washing and climbing the stairs. He has a past medical history of age-related macular degeneration, systolic heart failure (ejection fraction on echocardiogram = 43%), osteoporosis and myelodysplasia. He is currently taking ramipril, bisoprolol and spironolactone, at the maximum tolerated doses, as well as alendronate once weekly. Which medical therapy could further improve his prognosis?

- ○ Erythropoietin
- ○ Blood transfusion
- ○ Isosorbide mononitrate and hydralazine
- ○ Folic acid
- ○ Furosemide

Submit answer

Reference ranges

Score: **20.1%**

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Question 141 of 328 ✓

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Erythropoietin	10%
Blood transfusion	7%
Isosorbide mononitrate and hydralazine	67%
Folic acid	5%
Furosemide	11%

Hydrazine and nitrate should be considered for Afro-Caribbean patients with heart failure who are not responding to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Important for me Less important

This gentleman is symptomatic (New York Heart Association Stage III). He is fully optimised on conventional medical treatment, but NICE recommends that Afro-Caribbean patients are additionally treated with hydralazine and nitrate, to improve prognosis. Furosemide may improve his symptoms, but does not change his prognosis. This gentleman may also be symptomatic from anaemia, due to myelodysplasia, for which folic acid, erythropoietin and blood transfusion may improve his symptoms, but not his prognosis. The management of myelodysplasia in elderly patients with multiple co-morbidities is usually supportive care.

Discuss (4) Improve

Next question >

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

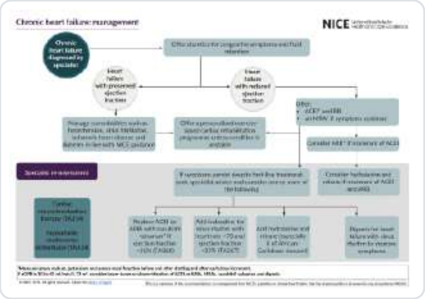
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death

- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
  - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments
- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
  - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure: management
NICE

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

[2012 Guidance on ivabradine]

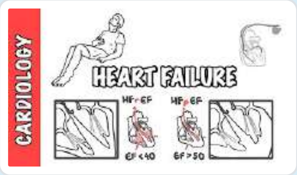
NICE 42 15

[2018 Chronic heart failure guidelines]

Suggest link

Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
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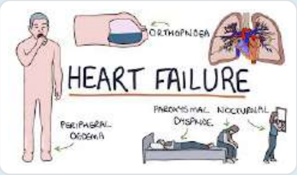
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


HEART FAILURE
ORTHOPNOEA
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **20.7%**

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Question 142 of 328

A 56-year-old Caucasian male patient with no history of ischaemic heart disease had a non-ST elevation myocardial infarction 20 days ago and was started on aspirin, atorvastatin, bisoprolol and enalapril as an inpatient. He has been discharged to your care in the cardiology outpatients department. He presents for review 1 month later saying he remains breathless on minimal exertion (walking 50 metres) with no chest pains. There is no evidence of pulmonary oedema on examination. A recent echocardiogram shows a left ventricular ejection fraction of 40%. His beta natriuretic peptide level is 436 pg/mL (normal <100 pg/mL).

What would be most appropriate to add to his oral therapy?

- ○ Furosemide ×
- ○ Spironolactone ×
- ○ Hydralazine and isosorbide dinitrate (modified release) ×
- ○ Irbesartan ×
- ○ Ivabradine ×

Submit answer

Reference ranges

Score: **20.7%**

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142	-

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Question 142 of 328 ✗

A 56-year-old Caucasian male patient with no history of ischaemic heart disease had a non-ST elevation myocardial infarction 20 days ago and was started on aspirin, atorvastatin, bisoprolol and enalapril as an inpatient. He has been discharged to your care in the cardiology outpatients department. He presents for review 1 month later saying he remains breathless on minimal exertion (walking 50 metres) with no chest pains. There is no evidence of pulmonary oedema on examination. A recent echocardiogram shows a left ventricular ejection fraction of 40%. His beta natriuretic peptide level is 436 pg/mL (normal <100 pg/mL).

What would be most appropriate to add to his oral therapy?

Furosemide	8%
Spironolactone	80%
Hydralazine and isosorbide dinitrate (modified release)	6%
Irbesartan	2%
Ivabradine	4%

NICE guidelines regarding chronic heart failure with reduced left ventricular ejection fraction:

- Start an ACE-inhibitor and β blocker in increasing doses to maximum tolerated.
- Second line treatment most appropriate for this Caucasian patient is spironolactone because of the recent history of myocardial infarction.
- Furosemide has no prognostic benefit and is not indicated if there are no signs of fluid overload on examination.
- Eplerenone is a mineralocorticoid antagonist that is specifically marketed for post-myocardial infarction patients. However, it is not available off-patent in the UK and therefore rarely first line on hospital formularies

👍 👎 Discuss (6) Improve

Next question >

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

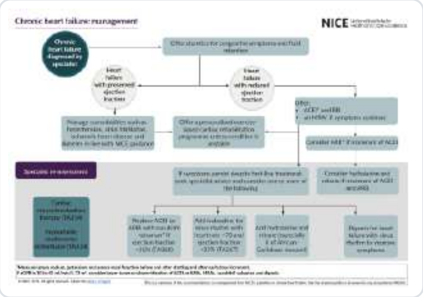
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
  - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
  - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

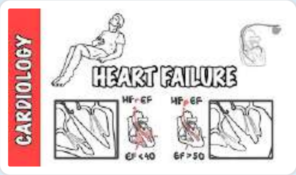
2012 Guidance on ivabradine

NICE 42 15

2018 Chronic heart failure guidelines

Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
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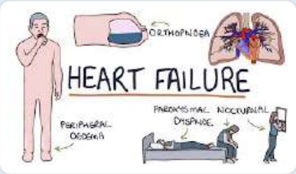
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1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Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

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Osmosis - YouTube 👍 0 👎 2

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Score: **20.6%**

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Back to top

A 44-year-old man attends with a 2-day history of fevers on rigours. On examination, you note splinter haemorrhages and a pan systolic murmur.

Blood results are as follows:

Hb	130 g/L	Male: (135-180) Female: (115 - 160)
Platelets	380 * 10^9/L	(150 - 400)
WBC	18.2 * 10^9/L	(4.0 - 11.0)
Na^+	126 mmol/L	(135 - 145)
K^+	4.4 mmol/L	(3.5 - 5.0)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	115 µmol/L	(55 - 120)
CRP	82 mg/L	(< 5)

Three sets of blood cultures are positive for the following organism:

Blood cultures	Gram +ve coagulase positive cocci

What treatment is indicated?

- ○ Benzylpenicillin and low-dose gentamicin
- ○ Flucloxacillin
- ○ Flucloxacillin, rifampicin and low-dose gentamicin
- ○ Vancomycin and low-dose gentamicin
- ○ Vancomycin and rifampicin

Submit answer

Reference ranges

Score: **20.6%**

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Creatinine	115 µmol/L	(55 - 120)
CRP	82 mg/L	(< 5)

Three sets of blood cultures are positive for the following organism:

Blood cultures	Gram +ve coagulase positive cocci

What treatment is indicated?

Benzylpenicillin and low-dose gentamicin	15%
Flucloxacillin	68%
Flucloxacillin, rifampicin and low-dose gentamicin	10%
Vancomycin and low-dose gentamicin	5%
Vancomycin and rifampicin	2%

IV flucloxacillin is the treatment of choice for native valve endocarditis caused by staphylococci

Important for me Less important

Flucloxacillin is correct. The positive cultures are highly suggestive of *S.aureus* infection due to the positive coagulase test result. *Streptococci* are not coagulase producing. Native valve endocarditis caused by *S.aureus* is treated with high-dose intravenous flucloxacillin.

Benzylpenicillin and low-dose gentamicin is incorrect. This combination of drugs is used to treat endocarditis caused by less sensitive *Streptococci*.

Flucloxacillin, rifampicin and low-dose gentamicin is incorrect. This combination of drugs is used to treat prosthetic valve endocarditis caused by *S.aureus*.

Vancomycin and low-dose gentamicin is incorrect. This combination of antibiotics can be used for initial blind therapy in patients with a penicillin allergy, a history of methicillin-resistant *Staphylococcus aureus* (MRSA) or severe sepsis.

Vancomycin and rifampicin is incorrect. This combination of antibiotics can be used for native valve endocarditis caused by *S.aureus* if they have a penicillin allergy or a history of MRSA.

Discuss (2) Improve

Next question

Infective endocarditis: prognosis and management

Poor prognostic factors

- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism

- staphylococci - 30%
- bowel organisms - 15%

- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin If prosthetic valve • vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA • vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA • vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic • vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin

Scenario	Suggested antibiotic therapy
	If penicillin allergic • vancomycin + low-dose gentamicin

Indications for surgery

- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

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143	✗

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A 27-year-old man presents to the emergency department. He was recently discharged following an internal fixation of his left tibia. The fracture occurred due to a fall whilst intoxicated and he broke his leg as he landed on the pavement.

He has returned because he experienced a sudden onset of shortness of breath and became concerned. He has no cough or chest pain. He has no previous medical problems. His medications include codeine and paracetamol; post-operative analgesia that he was discharged on. He denies any allergies.

Observations demonstrate hypoxia with saturations at 92% on room air and are otherwise unremarkable. Blood tests, including a FBC, U&E and CRP are all within normal ranges. Clinical examination reveals a few left-sided crepitations and there is swelling and tenderness of his left calf. This was the side of his operation. A chest X-ray demonstrates a small left-sided pleural effusion causing blunting of his left costophrenic angle. No CT or US scanning is available as it is 3 AM.

What is the most appropriate next step?

- ○ Treatment dose apixaban
- ○ Wait to morning then order CTPA
- ○ Immediate drainage of effusion
- ○ Wait to morning and then US guided drainage of effusion
- ○ Wait to morning then order VQ scan

Submit answer

Reference ranges

Question 144 of 328 ✗

A 27-year-old man presents to the emergency department. He was recently discharged following an internal fixation of his left tibia. The fracture occurred due to a fall whilst intoxicated and he broke his leg as he landed on the pavement.

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Observations demonstrate hypoxia with saturations at 92% on room air and are otherwise unremarkable. Blood tests, including a FBC, U&E and CRP are all within normal ranges. Clinical examination reveals a few left-sided crepitations and there is swelling and tenderness of his left calf. This was the side of his operation. A chest X-ray demonstrates a small left-sided pleural effusion causing blunting of his left costophrenic angle. No CT or US scanning is available as it is 3 AM.

What is the most appropriate next step?

Treatment dose apixaban	87%
Wait to morning then order CTPA	8%
Immediate drainage of effusion	3%
Wait to morning and then US guided drainage of effusion	2%
Wait to morning then order VQ scan	1%

Strong suspicion of PE but a delay in the scan: start on treatment dose anticoagulant meanwhile

Important for me Less important

The correct answer is treatment dose apixaban. This is a young and fit patient who has presented with sudden onset of shortness of breath with a swollen calf following immobilisation and surgery. The most likely diagnosis is a PE from a DVT. The clinical likelihood is very high and therefore immediate treatment is appropriate as the patient is hypoxic. Waiting to confirm the diagnosis should be done but it is more appropriate to start treatment first. The effusion is most likely secondary to an embolus and unlikely to be responsible for the level of hypoxia as it is small on the X-ray.

👍 👎 💬 Discuss (5) Improve

Next question ›

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:

- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

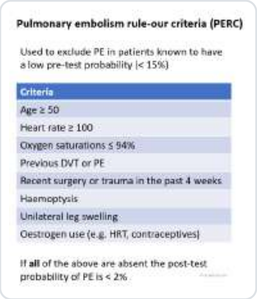
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
    - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5

Clinical feature	Points
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores

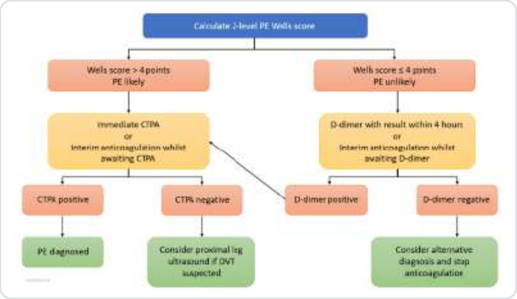
- PE likely - more than 4 points
- PE unlikely - 4 points or less

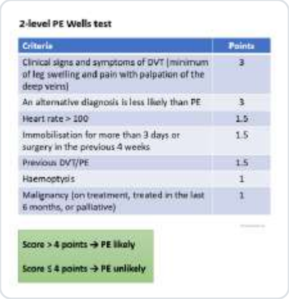
If a PE is 'likely' (more than 4 points)

- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
    - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
    - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points PE likely
Wells score ≤ 4 points PE unlikely
Immediate CTPA or Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours or Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

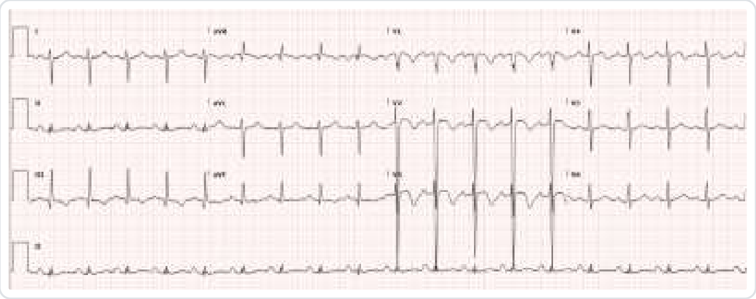
Some other points

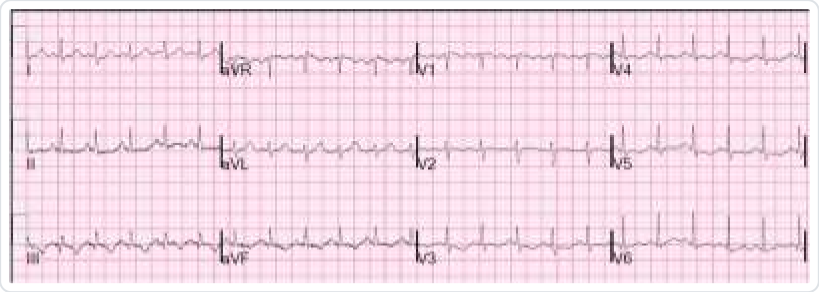
D-dimers

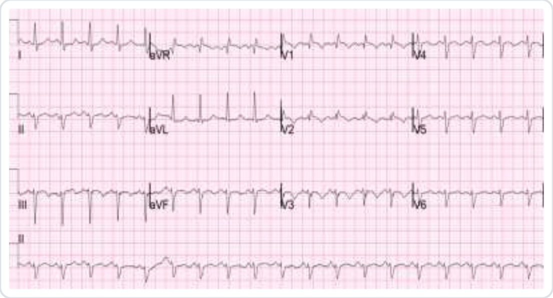
- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen







Chest x-ray

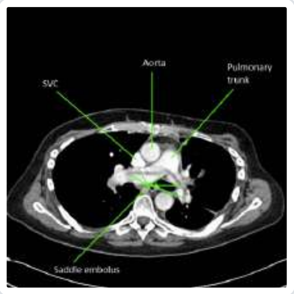
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

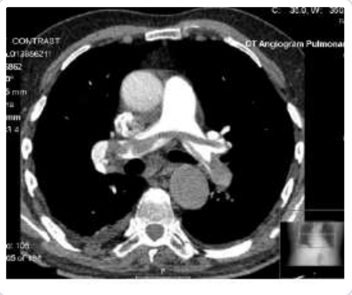
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus



1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 13 👎 10

The ECG in Pulmonary Embolism

MDcalc 👍 15 👎 8

Wells' Criteria for Pulmonary Embolism

MDcalc 👍 17 👎 8

PERC Rule for Pulmonary Embolism

NICE 👍 20 👎 9

2020 Venous thromboembolism guidelines

Suggest link

Report broken link

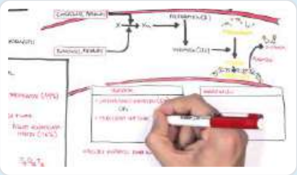
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

Report broken media

Score: **20.3%**

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Question 145 of 328

A 68-year-old male is admitted with crushing chest pain radiating to the left arm and an ECG suggestive of anterior ST-elevation myocardial infarction. He is treated under the local primary percutaneous cardiac intervention protocol and a drug-eluting stent is successfully deployed in his left anterior descending artery. A left ventriculogram is performed during the procedure indicating severely reduced systolic function. An echocardiogram confirms this with an ejection fraction calculated at 32%.

Which rate control medication would be the most appropriate first choice in treating this gentleman?

○ Carvedilol ×

○ Ivabradine ×

○ Metoprolol ×

○ Atenolol ×

○ Diltiazem ×

Submit answer

Reference ranges

Score: **20.3%**

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A 68-year-old male is admitted with crushing chest pain radiating to the left arm and an ECG suggestive of anterior ST-elevation myocardial infarction. He is treated under the local primary percutaneous cardiac intervention protocol and a drug-eluting stent is successfully deployed in his left anterior descending artery. A left ventriculogram is performed during the procedure indicating severely reduced systolic function. An echocardiogram confirms this with an ejection fraction calculated at 32%.

Which rate control medication would be the most appropriate first choice in treating this gentleman?

Carvedilol	52%
Ivabradine	14%
Metoprolol	19%
Atenolol	10%
Diltiazem	6%

Carvedilol, bisoprolol and nebivolol are the B-blockers that are licensed in the United Kingdom for the treatment of heart failure

Important for me Less important

This is a patient that has recently suffered a myocardial infarction with residual reduced ejection fraction secondary to that. The beta-blockers currently licensed in the treatment of heart failure and as secondary prevention of ischaemic cardiac disease include bisoprolol, carvedilol and nebivolol.

Ivabradine has a role in the management of angina and rate-control in patients with heart failure however it does not appear to be involved in the first line management and secondary prevention of ischaemic heart disease.

Diltiazem is a rate-limiting calcium channel blocker; all calcium channel blockers (rate limiting) are contraindicated in moderate to severe heart failure.

Metoprolol has previously shown increased rate of adverse events in early use in coronary artery disease (STEMI) while atenolol is not licensed in the UK for heart failure management.

👍 👎 💬 Discuss (4) Improve

Next question ›

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause

hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

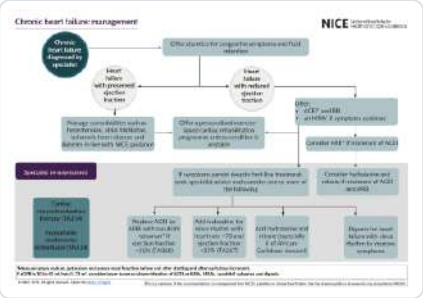
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
  - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**

    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years



Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

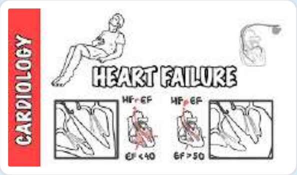
2012 Guidance on ivabradine

NICE 42 15

2018 Chronic heart failure guidelines

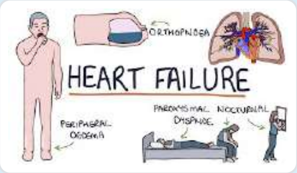
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PERIPHERAL OEDEMA
PAROXYSMAL NOCTURNAL DYSPNOEA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

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Score: **20.1%**

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A 62-year-old man sees his GP with complaints of leg pain. Over the last few months, he notices a crampy uncomfortable feeling in the back of both of his calves when he walks to the shops. The discomfort can be so bad that he has to stop and rest for a few minutes after which he notices that his symptoms are improved. On occasions, he also gets the symptoms when he is shopping in the supermarket. He attributes this to overexerting himself and tries to rest on his shopping trolley while walking but does not seem to help. He has a 40-pack year smoking history and takes amlodipine for his blood pressure, and paracetamol and ibuprofen for lower back pain that has troubled him for years.

Physical examination reveals mild atrophy of his thigh and calf muscles bilaterally, in addition to shiny pale skin with significant hair loss throughout his lower limbs. His pedal pulses are bilaterally impalpable, and popliteal pulses are faint. Power in both lower limbs is normal throughout all movements, and he has normal patellar reflexes bilaterally and absent ankle reflexes. His Babinski reflex is downgoing on the left side and equivocal on the right side. A recently obtained ankle brachial pressure index test yielded a result of 0.70 on the right side and 0.95 on the left side. X-rays of his lumbar spine show evidence of joint space narrowing and osteophytes.

Which one of the following is the next best step in the management of this patient?

○ MRI scan of lumbar spine

○ Check his HbA1c to screen for diabetes

○ Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery

○ Starting him on duloxetine to manage his pain

○ Screen for coronary artery disease with ECG stress testing

Submit answer

Reference ranges

Score: **20.1%**

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Question 146 of 328 ✗

A 62-year-old man sees his GP with complaints of leg pain. Over the last few months, he notices a crampy uncomfortable feeling in the back of both of his calves when he walks to the shops. The discomfort can be so bad that he has to stop and rest for a few minutes after which he notices that his symptoms are improved. On occasions, he also gets the symptoms when he is shopping in the supermarket. He attributes this to overexerting himself and tries to rest on his shopping trolley while walking but does not seem to help. He has a 40-pack year smoking history and takes amlodipine for his blood pressure, and paracetamol and ibuprofen for lower back pain that has troubled him for years.

Physical examination reveals mild atrophy of his thigh and calf muscles bilaterally, in addition to shiny pale skin with significant hair loss throughout his lower limbs. His pedal pulses are bilaterally impalpable, and popliteal pulses are faint. Power in both lower limbs is normal throughout all movements, and he has normal patellar reflexes bilaterally and absent ankle reflexes. His Babinski reflex is downgoing on the left side and equivocal on the right side. A recently obtained ankle brachial pressure index test yielded a result of 0.70 on the right side and 0.95 on the left side. X-rays of his lumbar spine show evidence of joint space narrowing and osteophytes.

Which one of the following is the next best step in the management of this patient?

MRI scan of lumbar spine	8%
Check his HbA1c to screen for diabetes	7%
Refer to vascular surgery for consideration of peripheral arterial stenting or bypass surgery	76%
Starting him on duloxetine to manage his pain	2%
Screen for coronary artery disease with ECG stress testing	8%

In patients with cardiovascular risk factors and symptoms suggestive of claudication with an equivocal/borderline ankle brachial pressure index study result, the next best study is an ankle brachial pressure index after exercise

Important for me Less important

This gentleman has symptoms of claudication, in particular, vascular claudication. Although he does have a history of chronic back pain for which he takes analgesia, he is less likely to have neurogenic claudication compared to vascular claudication. An important discriminating factor between neurogenic claudication and vascular claudication is that patients with neurogenic claudication have symptoms on exertion that improve with manoeuvers such as leaning forward, and sitting down, whereas vascular claudication does not change with these manoeuvers and only improves with rest. In addition, his physical exam shows evidence of peripheral arterial disease characterised with muscle atrophy, shiny skin with hair loss and impalpable pedal pulses. Absent ankle jerk reflexes and equivocal Babinski reflexes can be normal variants and do not always indicate neurological pathology. This patient has rightly already undergone an ankle brachial pressure index assessment, and the results indicate a diagnosis of peripheral arterial disease. He is functionally impaired by his symptoms and so the next best step in his management would be to refer him to vascular surgery for consideration of treatment strategies which may include percutaneous interventions with stenting and/or surgical bypass.

Although this patient has evidence of degenerative joint disease on his lumbar spine x-ray, his clinical presentation is not consistent with neurogenic claudication and so an MRI scan of his lumbar spine is not indicated.

Although it is important to screen for and aggressively manage cardiovascular risk factors in patients with peripheral arterial disease, this in itself would not address the patient's symptoms or the disease course.

This patient's pain is most consistent with vascular claudication as opposed to neuropathic pain, for which an agent like duloxetine would be appropriate.

Although individuals with peripheral arterial disease have a significant likelihood of having concomitant coronary disease, this patient is not complaining of angina or symptoms suggestive of coronary disease and so screening for this is not indicated. The next best step in managing this patient is to address and treat his symptoms related to his peripheral arterial disease.

Discuss (7) Improve

Next question >

Ankle-brachial pressure index

The ankle-brachial pressure index (ABPI) is the ratio of the systolic blood pressure in the lower leg to that in the arms. Lower blood pressure in the legs (result in a ABPI < 1) is an indicator of peripheral arterial disease (PAD). ABPI is therefore useful in evaluating patients with suspected PAD, for example a male smoker who presents with intermittent claudication.

It is also important to determine the ABPI in patients with leg ulcers. Venous ulcers are often treated with compression bandaging. Doing this in a patient with PAD could however be harmful as it would further restrict the blood supply to the foot. ABPIs should therefore always be measured in patients with leg ulcers.

Interpretation of ABPI
- > 1.2: may indicate calcified, stiff arteries. This may be seen with advanced age or PAD
- 1.0 - 1.2: normal
- 0.9 - 1.0: acceptable
- < 0.9: likely PAD. Values < 0.5 indicate severe disease which should be referred urgently

The ABPI is a good test, values less than 0.90 have been shown to have a sensitivity of 90% and a specificity of 98%* for PAD.

Compression bandaging is generally considered acceptable if the ABPI >= 0.8.

*Yao ST, Hobbs JT, Irvine WT. Ankle systolic pressure measurements in arterial disease affecting the lower extremities. Br J Surg. Sep 1969;56(9):676-9.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 3 👎 5

2010 Management of chronic venous leg ulcers

Suggest link

Report broken link

Score: **20%**

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Question 147 of 328

A 43-year-old man presents with palpitations to the emergency department. His ECG demonstrates a broad complex tachycardia at 180 beats/min, suggestive of ventricular tachycardia. His blood pressure is 134/80 mmHg, he is uncomfortable but alert. The emergency department doctor has administered a 300mg bolus of amiodarone and the consultant subsequently inserted a central venous line to continue amiodarone treatment with a 900mg infusion over 24 hours. He transiently settles but on the second day of admission, his cardiac monitor demonstrates persistent VT at 180 beats per minute again. His blood pressure is 140/75 mmHg. What is your treatment?

- ○ No treatment ×
- ○ Repeat bolus of amiodarone 300mg ×
- ○ DC cardioversion ×
- ○ Intravenous lidocaine infusion ×
- ○ Emergency VT ablation ×

Submit answer

Reference ranges

Score: **20%**

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A 43-year-old man presents with palpitations to the emergency department. His ECG demonstrates a broad complex tachycardia at 180 beats/min, suggestive of ventricular tachycardia. His blood pressure is 134/80 mmHg, he is uncomfortable but alert. The emergency department doctor has administered a 300mg bolus of amiodarone and the consultant subsequently inserted a central venous line to continue amiodarone treatment with a 900mg infusion over 24 hours. He transiently settles but on the second day of admission, his cardiac monitor demonstrates persistent VT at 180 beats per minute again. His blood pressure is 140/75 mmHg. What is your treatment?

No treatment	3%
Repeat bolus of amiodarone 300mg	20%
DC cardioversion	32%
Intravenous lidocaine infusion	38%
Emergency VT ablation	8%

The patient has entered refractory VT, continuing despite a therapeutic dose of amiodarone and sufficient loading. It is unlikely further boluses of amiodarone will produce additional therapeutic effects. Similarly, no treatment is not an option, his myocardium is unlikely to continue tolerating such a persistent tachycardia without soon reducing left ventricular output and arrest. He is not haemodynamically unstable and hence does not justify DC cardioversion as an emergency procedure. Similarly, emergency VT ablation is not appropriate after only 1st line treatment. Intravenous lidocaine is the second line treatment: first as a bolus, followed by an infusion regime over 6.5 hours.

Discuss (8) Improve

Next question

Ventricular tachycardia: management

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure, syncope) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks.

Drug therapy
- amiodarone: ideally administered through a central line
- lidocaine: use with caution in severe left ventricular impairment
- procainamide

Verapamil should NOT be used in VT.

If drug therapy fails
- electrophysiological study (EPS)
- implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 43 👎 7

2021 Tachycardia algorithm

Suggest link Report broken link

Score: **19.9%**

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Question 148 of 328

A 25-year-old female presents with palpations and dizziness. She states that she has had a few episodes similar to this in the past and each time they have spontaneously resolved. On examination she has a blood pressure of 85/65 mmHg and a heart rate of 180bpm. An ECG shows an irregularly irregular rhythm, no identifiable P waves, and a QRS of 135ms.

Your senior decides to electrically cardiovert her. While the cardioversion is being prepared you instruct the patient to perform a vagal manoeuvre. Shortly after this she loses consciousness and goes into cardiac arrest.

What is the most likely underlying disorder?

- ○ Torsades de Pointes ×
- ○ Atrial fibrillation ×
- ○ Atrioventricular nodal re-entry tachycardia (AVNRT) ×
- ○ Monomorphic ventricular tachycardia ×
- ○ Wolff-Parkinson-White syndrome (WPW) ×

Submit answer

Reference ranges

Score: **19.9%**

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148 -

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A 25-year-old female presents with palpations and dizziness. She states that she has had a few episodes similar to this in the past and each time they have spontaneously resolved. On examination she has a blood pressure of 85/65 mmHg and a heart rate of 180bpm. An ECG shows an irregularly irregular rhythm, no identifiable P waves, and a QRS of 135ms.

Your senior decides to electrically cardiovert her. While the cardioversion is being prepared you instruct the patient to perform a vagal manoeuvre. Shortly after this she loses consciousness and goes into cardiac arrest.

What is the most likely underlying disorder?

Torsades de Pointes	16%
Atrial fibrillation	10%
Atrioventricular nodal re-entry tachycardia (AVNRT)	16%
Monomorphic ventricular tachycardia	7%
Wolff-Parkinson-White syndrome (WPW)	51%

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation

Important for me   Less important

There are few arrhythmias, which can cause an irregularly irregular broad complex tachycardia. These include irregular atrial arrhythmias with bundle branch block (e.g. atrial fibrillation), and polymorphic ventricular tachycardia (including Torsades de Pointes). Another important group of conditions to be aware of include the pre-excitation syndromes.

Pre-excitation refers to early activation of the ventricles due to impulses bypassing the AV node via an accessory pathway. This results in a broad QRS complex e.g. the slurred upstroke of QRS (delta wave) as is seen in WPW. When present with atrial fibrillation, the accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node. This results in an irregularly irregular broad complex tachycardia.

The Valsalva manoeuvre increases the vagal tone resulting in AV node block. This promotes conduction of the atrial signals through the accessory pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF, as is seen in this case.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation. In a haemodynamically unstable patient, urgent synchronised DC cardioversion is required. Medical treatment options in a stable patient include procainamide, although DC cardioversion may be preferred. Procainamide is a class 1 anti-arrhythmic which preferentially blocks the accessory pathway.

👍 👎 💬 Discuss (9) Improve

Next question ›

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

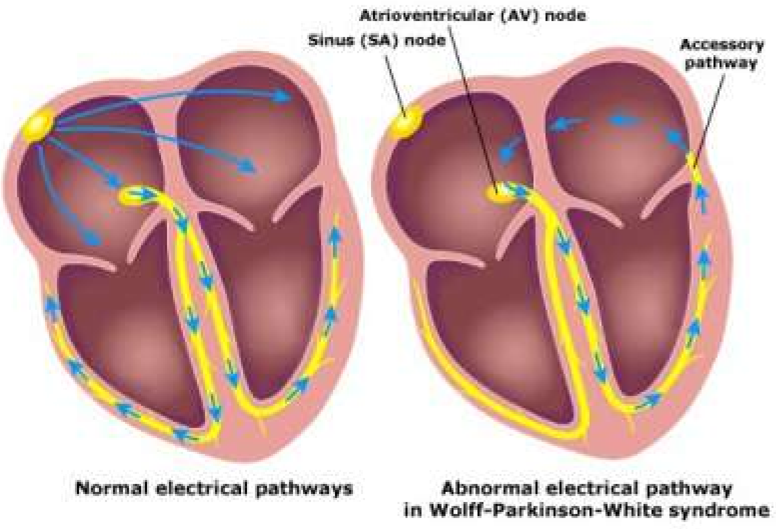
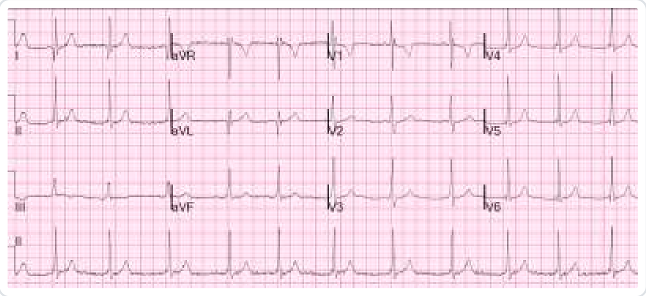

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
    - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway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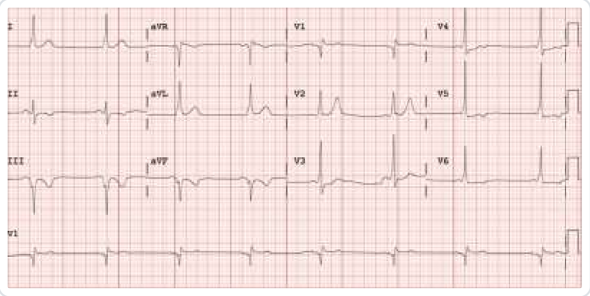
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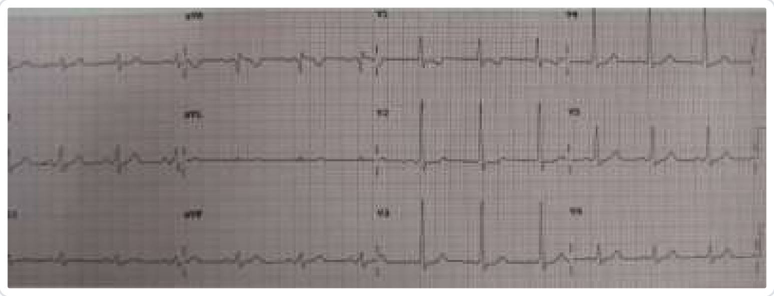
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Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
    - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 12 2

Report broken media

Score: **19.7%**

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A 67-year-old woman is reviewed in cardiology clinic following a GP referral. A recent transthoracic echocardiogram demonstrated severe aortic stenosis with an aortic valve at 0.7cm2 and a mean pressure gradient of 62mmHg and a left ventricular ejection fraction of 43%. Pulmonary arterial pressure is normal. She has a chest X-ray two months ago which was normal. She denies any symptoms of shortness of breath, chest pain or lightheadedness.

She is normally fit and well with a past medical history of hypothyroidism, two caesarian sections, osteoarthritis and migraines. She is a non-smoker. What is the most appropriate management plan?

- ○ Review in six months ×
- ○ Cardiac MRI ×
- ○ Exercise test ×
- ○ Aortic valve replacement ×
- ○ Transcatheter aortic valve implantation (TAVI) ×

Submit answer

Reference ranges

Score: **19.7%**

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149 -

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A 67-year-old woman is reviewed in cardiology clinic following a GP referral. A recent transthoracic echocardiogram demonstrated severe aortic stenosis with an aortic valve at 0.7cm2 and a mean pressure gradient of 62mmHg and a left ventricular ejection fraction of 43%. Pulmonary arterial pressure is normal. She has a chest X-ray two months ago which was normal. She denies any symptoms of shortness of breath, chest pain or lightheadedness.

She is normally fit and well with a past medical history of hypothyroidism, two caesarian sections, osteoarthritis and migraines. She is a non-smoker. What is the most appropriate management plan?

Review in six months	17%
Cardiac MRI	2%
Exercise test	2%
Aortic valve replacement	56%
Transcatheter aortic valve implantation (TAVI)	22%

The correct answer is aortic valve replacement. This is an asymptomatic patient with severe stenosis but she has an ejection fraction of less than 50%. This means that she should be referred for aortic valve replacement or TAVI if unsuitable. Exercise testing would be recommended if her ejection fraction was greater than 50%. Cardiac MRI would not be helpful in this instance. If she had an ejection fraction greater than 50% and passed exercise testing then she could be reviewed in six months.

Source:
Vahanian A., Alfieri O., Andreotti F., et al. Guidelines on the management of valvular heart disease (version 2012). The Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery. European Heart Journal. 2012;33:24512496. doi: 10.1093/eurheartj/ehs109

👍 👎 Discuss (15) Improve

Next question >

Aortic stenosis

Clinical features of symptomatic disease

- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur
    - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
    - classically radiates to the carotids
    - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement

- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
  - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
  - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
  - may be used in children with no aortic valve calcification
  - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

AI + Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

Guidelines on the management of valvular heart disease

Suggest link Report broken link

Media

Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1

AORTIC
STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

AORTIC VALVE
DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2

Report broken media

Score: **19.6%**

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Question 150 of 328

A 62-year-old woman is reviewed prior to discharge by the medical team. She was admitted with a lower respiratory tract infection. She now feels well and is ready for discharge. She has a background of type two diabetes mellitus, hypertension, hypothyroidism, osteoarthritis and dementia. Her medication history includes metformin 500mg BD, ramipril 2.5mg OD, levothyroxine 75 mcg OD and paracetamol 500mg QDS PRN. On the first two days in hospital, her metformin was stopped, but her other medications continued as in the community during her admission.

It is noted that she has persistently high blood pressure despite being confirmed as taking ramipril. What is the most appropriate plan to control her hypertension?

- ○ Increase the dose of ramipril and check U&Es within 1-2 weeks
- ○ Increase the dose of ramipril and check U&Es within 3-4 weeks
- ○ Add amlodipine, increase the dose of ramipril and check U&Es within 3-4 weeks
- ○ Add on amlodipine at a low dose
- ○ Stop ramipril and start amlodipine instead

Submit answer

Reference ranges

Score: **19.6%**

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Back to top

Question 150 of 328 ✗

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Increase the dose of ramipril and check U&Es within 1-2 weeks	58%
Increase the dose of ramipril and check U&Es within 3-4 weeks	15%
Add amlodipine, increase the dose of ramipril and check U&Es within 3-4 weeks	7%
Add on amlodipine at a low dose	20%
Stop ramipril and start amlodipine instead	1%

The correct answer is to increase the dose of ramipril and to check U&Es within 1-2 weeks. The patient is on a low dose of ramipril and there is room to increase it, and this should be done prior to adding on a second anti-hypertensive. It is important to test renal function within two weeks of either starting or increasing the dose of an ACE inhibitor to exclude deteriorating renal function, which would need to be investigated promptly. Amlodipine would have been a more appropriate anti-hypertensive choice if she did not have diabetes.

Discuss (8) Improve

Next question >

ACE inhibitors ★

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in the secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibits the conversion angiotensin I to angiotensin II
  - → decrease in angiotensin II levels → to vasodilation and reduced blood pressure
  - → decrease in angiotensin II levels → reduced stimulation for aldosterone release → decrease in sodium and water retention by the kidneys
- renoprotective mechanism
  - angiotensin II constricts the efferent glomerular arterioles
  - ACE inhibitors therefore lead to dilation of the efferent arterioles → reduced glomerular capillary pressure → decreased mechanical stress on the delicate filtration barriers of the glomeruli
  - this is particularly important in diabetic nephropathy
- ACE inhibitors are activated by phase 1 metabolism in the liver

Side-effects:

- cough
  - occurs in around 15% of patients and may occur up to a year after starting treatment
  - thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications

- pregnancy and breastfeeding - avoid
- renovascular disease - may result in renal impairment
- aortic stenosis - may result in hypotension

- hereditary of idiopathic angioedema
- specialist advice should be sought before starting ACE inhibitors in patients with a potassium >= 5.0 mmol/L

Interactions

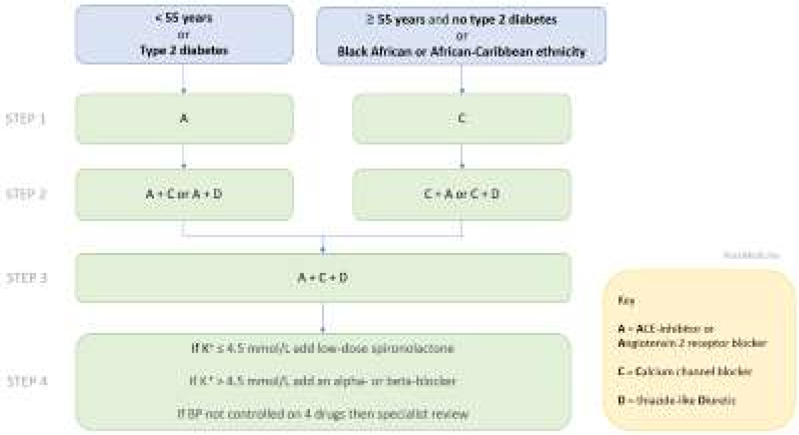
- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day)
  - significantly increases the risk of hypotension

Monitoring

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
  - a rise in the creatinine and potassium may be expected after starting ACE inhibitors
  - acceptable changes are an increase in serum creatinine, up to 30% from baseline and an increase in potassium up to 5.5 mmol/l.
  - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis

Examples of common ACE inhibitors

- ramipril
- enalapril
- lisinopril


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 53 👎 34

2014 Chronic kidney disease guidelines

BNF 👍 50 👎 25

ACE inhibitors

Suggest link Report broken link

Score: **19.5%**

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Question 151 of 328

A 56-year-old gentleman is admitted to Emergency Department with back pain. His past medical history included type 2 diabetes and hypertension. Initial observations are as follows: heart rate 112 beats per minute, blood pressure 155/82 mmHg, respiratory rate 26/min, oxygen saturations 95% on 2 litres oxygen via nasal cannula, temperature 37.2ºC.

Examination demonstrates muffled heart sounds I and II. JVP is not elevated and there is no peripheral oedema. Auscultation of the chest is clear with no crackles or wheeze and good air entry bilaterally. An ECG confirms sinus tachycardia with a heart rate of 102 beats per minute and 2mm inferior ST depression. Portable chest x-ray shows poor inspiratory effort with cardiomegaly and clear lung fields.

What single investigation would you arrange next?

- ○ CT aortogram ×
- ○ CT pulmonary angiogram (CTPA) ×
- ○ Ventilation/Perfusion scan (V/Q scan) ×
- ○ Echocardiogram ×
- ○ 12 hour troponin ×

Submit answer

Reference ranges

Score: **19.5%**

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What single investigation would you arrange next?

✓ CT aortogram	54%
✗ CT pulmonary angiogram (CTPA)	11%
Ventilation/Perfusion scan (V/Q scan)	1%
Echocardiogram	31%
12 hour troponin	3%

This patient has clinical features suggestive of type A aortic dissection, and an urgent CT aortogram would be required to exclude this diagnosis. Patients often describe back pain as the aorta is a retroperitoneal structure. A major risk factor is hypertension. The Stanford classification outlines type A dissections which involves the ascending aorta and type B dissections which originate in the descending aorta. Proximal extension of type A dissections can result in rupture of the coronary sinus and secondary ischaemia. They may be associated with pericardial effusions, which often manifests as cardiomegaly on a plain chest x-ray and results in muffled heart sounds. Management of type A dissections involves urgent referral to a cardiothoracic surgical unit.

The presentation in this example is not typical for a pulmonary embolus (PE), and therefore, CTPA or V/Q scan would not be beneficial. A 12-hour troponin would provide some useful information relating to myocardial necrosis but is not the single best investigation in this clinical context. An echocardiogram would be relevant to exclude a significant pericardial effusion with associated features of cardiac tamponade but the examination findings are not suggestive of this. Visualisation of proximal dissections involving the aortic root is feasible with echocardiography but the definitive test to establish diagnosis would be CT aortography.

👍 👎 Discuss (18) Improve

Next question >

Aortic dissection: features and classification ★

Aortic dissection is a rare but serious cause of chest pain.

Pathophysiology
- tear in the tunica intima of the wall of the aorta

Associations
- hypertension: the most important risk factor
- trauma
- bicuspid aortic valve
- collagens: Marfan's syndrome, Ehlers-Danlos syndrome
- Turner's and Noonan's syndrome
- pregnancy
- syphilis

Features:
- chest/back pain
    - typically severe and 'sharp', 'tearing' in nature
    - pain is typically maximal at onset
    - classically chest pain is more common in type A dissection and upper back pain is more common in type B dissection. However, there is considerable overlap and both chest and back pain are present in many patients

- pulse deficit
    - weak or absent carotid, brachial, or femoral pulse
    - variation (>20 mmHg) in systolic blood pressure between the arms
- aortic regurgitation
- hypertension
- other features may result from the involvement of specific arteries. For example:
    - coronary arteries → angina
    - spinal arteries → paraplegia
    - distal aorta → limb ischaemia
- the majority of patients have no or non-specific ECG changes. In a minority of patients, ST-segment elevation may be seen in the inferior leads

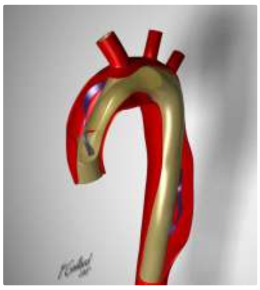
Classification

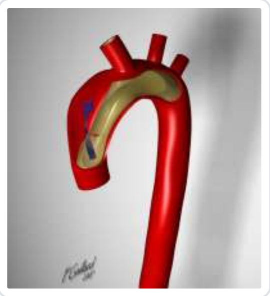
Stanford classification

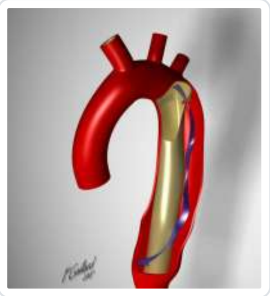
- type A - ascending aorta, 2/3 of cases
- type B - descending aorta, distal to left subclavian origin, 1/3 of cases

DeBakey classification

- type I - originates in ascending aorta, propagates to at least the aortic arch and possibly beyond it distally
- type II - originates in and is confined to the ascending aorta
- type III - originates in descending aorta, rarely extends proximally but will extend distally







123

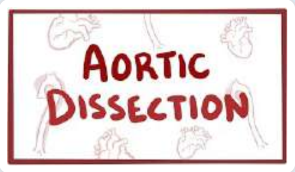
Next question

Textbooks

High-yield textbook

Extended textbook

Media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Aortic dissection

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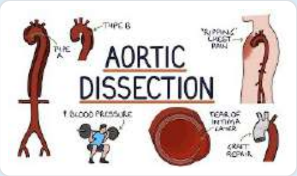
AORTIC
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TYPE B
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"RIPPING" CHEST PAIN
↑ BLOOD PRESSURE
TEAR OF INTIMA LAYER
GRAFT REPAIR

Understanding Aortic Dissection

Zero To Finals - YouTube 👍 1 👎 1

Report broken media

Score: **19.3%**

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A 65-year-old man is reviewed in the outpatient cardiology clinic and reports palpitations and fatigue when carrying out normal household tasks. He has a history of heart failure (with an ejection fraction of 41%) and chronic obstructive pulmonary disease. He takes bisoprolol 10mg once daily, ramipril 10mg once daily and uses a tiotropium bromide inhaler.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 145/82 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air (95% on exertion)

On examination, the pulse is regular.

What is the most appropriate next step in the patient's management?

○ Add a long-acting beta agonist and inhaled corticosteroid ×

○ Add digoxin ×

○ Add ivabradine ×

○ Add spironolactone ×

○ Increase the dose of bisoprolol ×

Submit answer

Reference ranges

Score: **19.3%**

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What is the most appropriate next step in the patient's management?

Add a long-acting beta agonist and inhaled corticosteroid	4%
Add digoxin	6%
Add ivabradine	18%
Add spironolactone	68%
Increase the dose of bisoprolol	5%

Offer a mineralcorticoid receptor antagonist, in addition to an ACE inhibitor (or ARB) and beta-blocker, to people who have heart failure with reduced ejection fraction if they continue to have symptoms of heart failure

Important for me Less important

Add spironolactone is correct. The patient reports palpitations and fatigue on minimal exertion. These symptoms suggest symptomatic heart failure rather than worsening COPD. This is reaffirmed by the normal SpO2 results following exertion. In a patient that is on maximally tolerated doses of beta-blocker and an ACE inhibitor, the correct next step is to add spironolactone.

Add a long-acting beta-agonist and inhaled corticosteroid is incorrect. Symptoms of palpitations and fatigue are more suggestive of symptomatic heart failure rather than COPD where wheezing and shortness of breath would be more typical. Therefore adjusting his COPD inhaler regimen is incorrect.

Add digoxin is incorrect. Digoxin is indicated in the management of symptomatic heart failure (especially in the context of co-existent atrial fibrillation) but only after other medications such as spironolactone have been initially trialled. This patient is not in AF.

Add ivabradine is incorrect. This is a 3rd line option for those patients with an ejection fraction < 35% and a heart rate > 75.

Increase the dose of bisoprolol is incorrect. 10mg is the maximum dose of bisoprolol that is recommended for heart failure.

👍 👎 Discuss (4) Improve

Next question >

Chronic heart failure: drug management

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**
- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

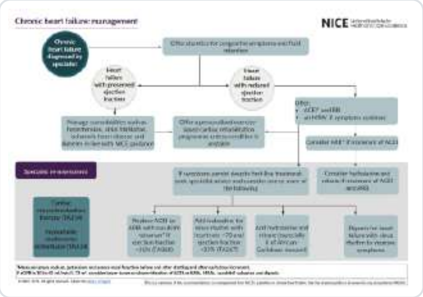
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period

- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

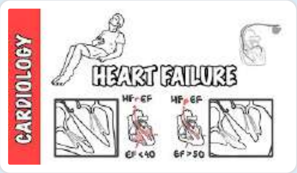
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

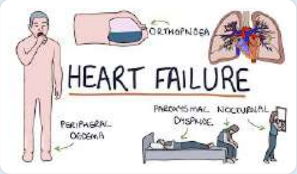
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **19.2%**

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Question 153 of 328

A 24-year-old female is brought into the emergency department by ambulance. She has a family history of Wolff-Parkinson-White syndrome and was binge drinking last night. She is complaining of palpitations which she says started suddenly 1 hour ago.

Her observations are as follows

- Heart rate 180 bpm
- Blood pressure 100/60 mmHg
- Saturations 98% on air
- Respiratory rate 26/min
- Temperature 36.8ºC

An ECG (electrocardiogram) is done which shows pre-excited atrial fibrillation (AF)

Which of the following is an appropriate treatment?

○ Adenosine ×

○ Atenolol ×

○ Flecainide ×

○ Diltiazem ×

○ Digoxin ×

Submit answer

Reference ranges

Score: **19.2%**

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152 ✗

153 -

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A 24-year-old female is brought into the emergency department by ambulance. She has a family history of Wolff-Parkinson-White syndrome and was binge drinking last night. She is complaining of palpitations which she says started suddenly 1 hour ago.

Her observations are as follows

- Heart rate 180 bpm
- Blood pressure 100/60 mmHg
- Saturations 98% on air
- Respiratory rate 26/min
- Temperature 36.8ºC

An ECG (electrocardiogram) is done which shows pre-excited atrial fibrillation (AF)

Which of the following is an appropriate treatment?

Adenosine	6%
Atenolol	9%
Flecainide	77%
Diltiazem	5%
Digoxin	3%

In pre-excited AF don't give anything that blocks conduction at AV node (including calcium channel blockers, adenosine or digoxin) as this can cause ventricular tachycardia

Important for me Less important

Flecainide is the only appropriate treatment on this list

All of the other options are drugs which block transmission through the

AV (atrioventricular) node. Giving these drugs in pre-excited AF (atrial fibrillation) can precipitate VT (ventricular tachycardia) or VF (ventricular fibrillation).

Note that flecainide is contraindicated in patients with structural heart abnormalities. In this situation then amiodarone can be given instead.

If any of the following are present: hypotension, signs of shock, altered mental status, chest pain or acute heart failure then synchronised cardioversion should be used rather than medication.

👍 | 👎 | Discuss (6) | Improve

Next question >

Atrial fibrillation: pharmacological cardioversion

★

NICE published guidelines on the management of atrial fibrillation (AF) in 2014. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2016 guidelines

Agents with proven efficacy in the pharmacological cardioversion of atrial fibrillation
- amiodarone
- flecainide (if no structural heart disease)
- others (less commonly used in UK): quinidine, dofetilide, ibutilide, propafenone

Less effective agents
- beta-blockers (including sotalol)
- calcium channel blockers
- digoxin
- disopyramide
- procainamide

HTTPS://T.ME/+AOWEXXQPZDM1NJZK

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 5

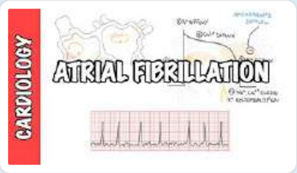
2014 NICE guidelines

European Society of Cardiology 👍 3 👎 3

2020 Atrial fibrillation guidelines

Suggest link Report broken link

Media


CARDIOLOGY
ATRIAL FIBRILLATION

Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 1 👎 0

Atrial fibrillation

Osmosis - YouTube 👍 1 👎 0

Report broken media

Score: **19.1%**

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A 48-year-old female presents with chest pain. She describes the pain as central, dull and tight. She has associated nausea. She has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	130ms
QRS	115ms
ST segments	ST elevation in V1-V3

Blood results are as follows:

Troponin (on admission)	< 0.015 µg/l (normal < 0.015)
Troponin (6 hours later)	< 0.015 µg/l (normal < 0.015)

What is the most likely diagnosis?

○ Acute STEMI

○ Acute NSTEMI

○ Unstable angina

○ Stable angina

○ Prinzmetal's angina (variant angina)

Submit answer

Reference ranges

A 48-year-old female presents with chest pain. She describes the pain as central, dull and tight. She has associated nausea. She has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	130ms
QRS	115ms
ST segments	ST elevation in V1-V3

Blood results are as follows:

Troponin (on admission)	< 0.015 µg/l (normal < 0.015)
Troponin (6 hours later)	< 0.015 µg/l (normal < 0.015)

What is the most likely diagnosis?

Acute STEMI	11%
Acute NSTEMI	2%
Unstable angina	15%
Stable angina	3%
Prinzmetal's angina (variant angina)	69%

In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis

Important for me Less important

The presence of ST elevation and chest pain should alert you to the diagnosis of acute STEMI. However importantly the serial troponins are negative effectively ruling out myocardial infarction. In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis. Prinzmetal's angina is therefore the most likely diagnosis.

Acute NSTEMI is excluded by the normal serial troponins.

Angina pectoris (stable or unstable) is of course a possibility. However the presence of ST elevation, the relatively young age of the patient, and the absence of cardiovascular risk factors, makes Prinzmetal's angina the more likely diagnosis.

The patient will need coronary angiography. The gold standard test for diagnosing variant angina is to visualise the coronary arteries by angiography before and after injection of a provocative agent such as acetylcholine to precipitate an attack of vasospasm.

👍 👎 Discuss (6) Improve

Next question >

ECG: ST elevation ★

Causes of ST elevation
- myocardial infarction
- pericarditis/myocarditis
- normal variant - 'high take-off'
- left ventricular aneurysm
- Prinzmetal's angina (coronary artery spasm)
- Takotsubo cardiomyopathy
- rare: subarachnoid haemorrhage

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **19%**

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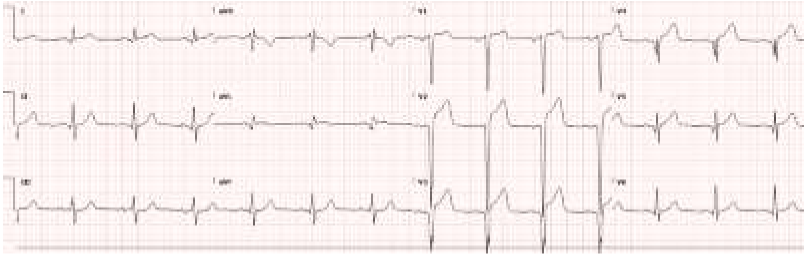
Back to top

A 72-year-old man arrives at the emergency room complaining of chest pain for six hours. It is a crushing pain at the centre of his chest, which radiates to the neck. He was diagnosed with Grave's disease previously, which was treated with radioiodine ablation, following which he developed hypothyroidism. He suffered an ischemic stroke two months ago. His medications include aspirin, atorvastatin and thyroxine. His heart rate is 83 beats/min, BP 130/80 mmHg, respiratory rate 20 breaths/min, and oxygen saturation is 98%. An ECG is taken:



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What is the most appropriate next step in managing this patient?

- ○ Give aspirin and arrange for an emergency CRP, troponin level and ECHO
- ○ Give aspirin and arrange for an emergency troponin level and ECHO
- ○ Give aspirin and glyceryl trinitrate and arrange for an emergency troponin level
- ○ Give aspirin and glyceryl trinitrate, and prepare for urgent PCI
- ○ Give aspirin and glyceryl trinitrate, and prepare for urgent fibrinolysis

Submit answer

Reference ranges ⌵

Score: **19%**

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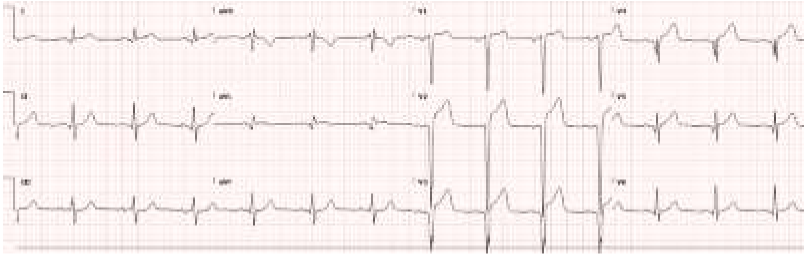
A 72-year-old man arrives at the emergency room complaining of chest pain for six hours. It is a crushing pain at the centre of his chest, which radiates to the neck. He was diagnosed with Grave's disease previously, which was treated with radioiodine ablation, following which he developed hypothyroidism. He suffered an ischemic stroke two months ago. His medications include aspirin, atorvastatin and thyroxine. His heart rate is 83 beats/min, BP 130/80 mmHg, respiratory rate 20 breaths/min, and oxygen saturation is 98%. An ECG is taken:



© Image used on license from Dr Smith, University of Minnesota

What is the most appropriate next step in managing this patient?

- Give aspirin and arrange for an emergency CRP, troponin level and ECHO 3%
- Give aspirin and arrange for an emergency troponin level and ECHO 4%
- Give aspirin and glyceryl trinitrate and arrange for an emergency troponin level 9%
- Give aspirin and glyceryl trinitrate, and prepare for urgent PCI 78%
- Give aspirin and glyceryl trinitrate, and prepare for urgent fibrinolysis 6%

The most appropriate next step is to **give aspirin and glyceryl trinitrate and plan for PCI**. The ECG in this scenario indicates a STEMI, with ST elevation observable in leads V1 to V5, which typically indicates an occlusion of the LAD. It is important to note that ECG manifestations of STEMI can vary depending on the timing post-ischemia onset. Initially, hyperacute T waves may be observed, followed by ST-segment elevation. As the condition evolves over several hours to days, T wave inversions and Q waves tend to appear within the same leads. Prompt primary PCI is essential for this patient in order to achieve reperfusion as soon as possible. Detailed management guidelines for acute coronary syndromes such as STEMI, NSTEMI, and unstable angina are outlined in the accompanying notes.

Give aspirin and arrange for an emergency CRP, troponin level, and ECHO is incorrect. Acute pericarditis is a close mimic of STEMI. However, it can be differentiated based on clinical features and ECG findings. Clinically, patients with pericarditis present with a sudden onset of a sharp pain over the anterior chest wall, similar to pleuritic pain, which increases on inspiration and decreases on leaning forwards. ECG findings include global ST elevations and PR-segment depressions in most limb leads (I, II, III, aVL, aVF) and precordial leads (V2 - V6). Initial investigations for pericarditis include CRP to check for infection, an ECHO scan to look for associated pericardial effusion and a troponin level. However, the history and ECG in this case do not suggest pericarditis.

Give aspirin and arrange for an emergency troponin level and ECHO is not appropriate in this scenario. This patient has typical features of STEMI in the ECG and should be taken up for PCI immediately. Troponin level and ECHO will be requested in all cases of myocardial infarction, but they will take time. Given that the patient has a STEMI, preparing for PCI is the most appropriate strategy here.

Giving aspirin and glyceryl trinitrate and arranging for an emergency troponin level would be misguided. In instances where STEMI has been identified by ECG findings'as it has been in this case'immediate reperfusion strategies such as PCI or fibrinolysis are warranted without delay. Troponin levels might still fall within normal ranges early after an acute coronary syndrome has occurred; therefore, they should not be solely relied upon for diagnosing STEMI.

Giving aspirin and glyceryl trinitrate and preparing for urgent fibrinolysis would be contraindicated due to the patient's recent history of ischemic stroke within the past three months. Fibrinolysis could potentially be considered for patients with no contraindications when they present with STEMI within 12 hours from symptom onset; however, it is only recommended if primary PCI is not accessible at their presenting hospital or if transfer to another facility offering PCI services cannot occur within 120 minutes.

👍 👎 Discuss (3) Improve

Next question >

Acute coronary syndrome: initial management

★

Acute coronary syndrome (ACS) is a very common and important presentation in medicine. The management of ACS has evolved over recent years, with the development of new drugs and procedures such as percutaneous coronary intervention (PCI).

Emergency departments often have their own protocols based on local factors such as the availability of PCI and hospital drug formularies. The following is based on the 2020 update to the NICE ACS guidelines.

Acute coronary syndrome can be classified as follows:

- ST-elevation myocardial infarction (STEMI): ST-segment elevation + elevated biomarkers of myocardial damage
- non ST-elevation myocardial infarction (NSTEMI): ECG changes but no ST-segment elevation + elevated biomarkers of myocardial damage
- unstable angina

The management of ACS depends on the particular subtype. NICE management guidance groups the patients into two groups:

- 1. STEMI
- 2. NSTEM/unstable angina

Common management of all patients with ACS

Initial drug therapy

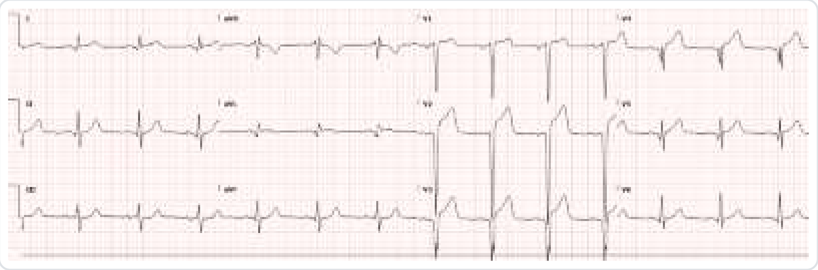
- aspirin 300mg
- oxygen should only be given if the patient has oxygen saturations < 94% in keeping with British Thoracic Society oxygen therapy guidelines

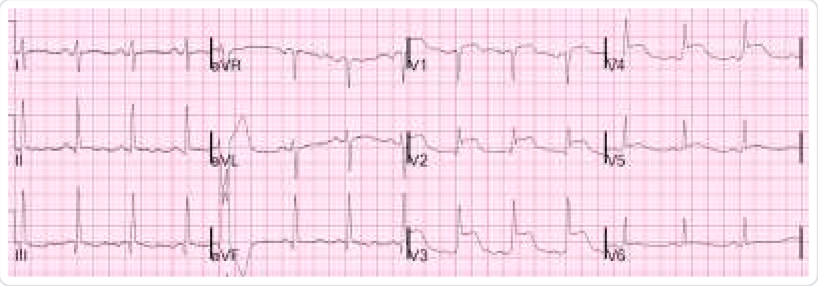
- morphine should only be given for patients with severe pain
  - previously IV morphine was given routinely
  - evidence, however, suggests that this may be associated with adverse outcomes
- nitrates
  - can be given either sublingually or intravenously
  - useful if the patient has ongoing chest pain or hypertension
  - should be used in caution if patient hypotensive

The next step in managing a patient with suspected ACS is to determine whether they meet the ECG criteria for STEMI. It is, of course, important to recognise that these criteria should be interpreted in the context of the clinical history.

STEMI criteria

- clinical symptoms consistent with ACS (generally of ≥ 20 minutes duration) with persistent (> 20 minutes) ECG features in ≥ 2 contiguous leads of:
  - 2.5 mm (i.e ≥ 2.5 small squares) ST elevation in leads V2-3 in men under 40 years, or ≥ 2.0 mm (i.e ≥ 2 small squares) ST elevation in leads V2-3 in men over 40 years
  - 1.5 mm ST elevation in V2-3 in women
  - 1 mm ST elevation in other leads
  - new LBBB (LBBB should be considered new unless there is evidence otherwise)





Management of STEMI

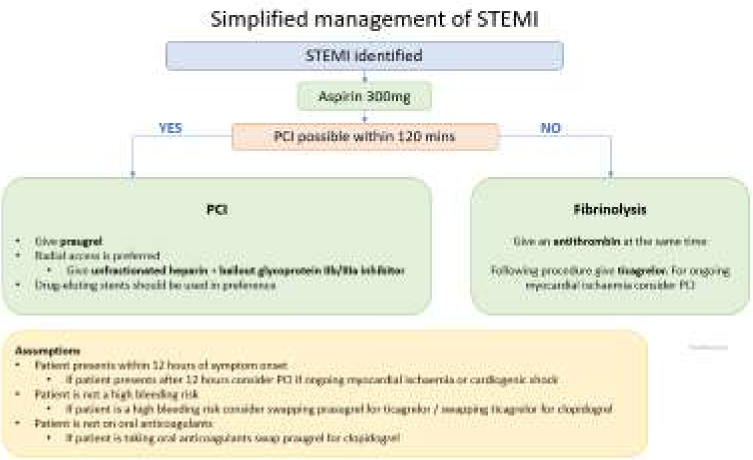

Simplified management of STEMI
STEMI identified
Aspirin 300mg
YES
PCI possible within 120 mins
NO
PCI
Give prasugrel
Radial access is preferred
Give unfractionated heparin + bailout glycoprotein IIb/IIIa inhibitor
Drug-eluting stents should be used in preference
Fibrinolysis
Give an antithrombin at the same time
Following procedure give ticagrelor. For ongoing myocardial ischaemia consider PCI
Assumptions
Patient presents within 12 hours of symptom onset
If patient presents after 12 hours consider PCI if ongoing myocardial ischaemia or cardiogenic shock
Patient is not a high bleeding risk
If patient is a high bleeding risk consider swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel for clopidogrel

Diagram showing the simplified management of STEMI according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

Once a STEMI has been confirmed the first step is to immediately assess eligibility for coronary reperfusion therapy. There are two types of coronary reperfusion therapy:

- percutaneous coronary intervention
  - should be offered if the presentation is within 12 hours of the onset of symptoms AND PCI can be delivered within 120 minutes of the time when fibrinolysis could have been given (i.e. consider fibrinolysis if there is a significant delay in being able to provide PCI)
  - if patients present after 12 hours and still have evidence of ongoing ischaemia then PCI should still be considered
  - drug-eluting stents are now used. Previously 'bare-metal' stents were sometimes used but have higher rates of restenosis
  - radial access is preferred to femoral access
- fibrinolysis
  - should be offered within 12 hours of the onset of symptoms if primary PCI cannot be delivered within 120 minutes of the time when fibrinolysis could have been given
  - a practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) that does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI

If patients are eligible this should be offered as soon as possible.

Percutaneous coronary intervention for patients with STEMI

Further antiplatelet prior to PCI

- this is termed 'dual antiplatelet therapy', i.e. aspirin + another drug
- if the patient is not taking an oral anticoagulant: prasugrel
- if taking an oral anticoagulant: clopidogrel

Drug therapy during PCI

- patients undergoing PCI with radial access:
  - unfractionated heparin with bailout glycoprotein IIb/IIIa inhibitor (GPI) - this is the action of using a GPI during the procedure when it was not intended from the outset, e.g. because of worsening or persistent thrombus
- patients undergoing PCI with femoral access:
  - bivalirudin with bailout GPI

Other procedures during PCI

- thrombus aspiration, but not mechanical thrombus extraction, should be considered
- complete revascularisation should be considered for patients with multivessel coronary artery disease without cardiogenic shock

Fibrinolysis for patients with STEMI

Fibrinolysis used to be the only form of coronary reperfusion therapy available. However, it is used much less commonly now given the widespread availability of PCI.

The contraindications to fibrinolysis and other factors are described in other notes.

Patients undergoing fibrinolysis should also be given an antithrombin drug.

An ECG should be repeated after 60-90 minutes to see if the ECG changes have resolved. If patients have persistent myocardial ischaemia following fibrinolysis then PCI should be considered.

Management of NSTEMI/unstable angina

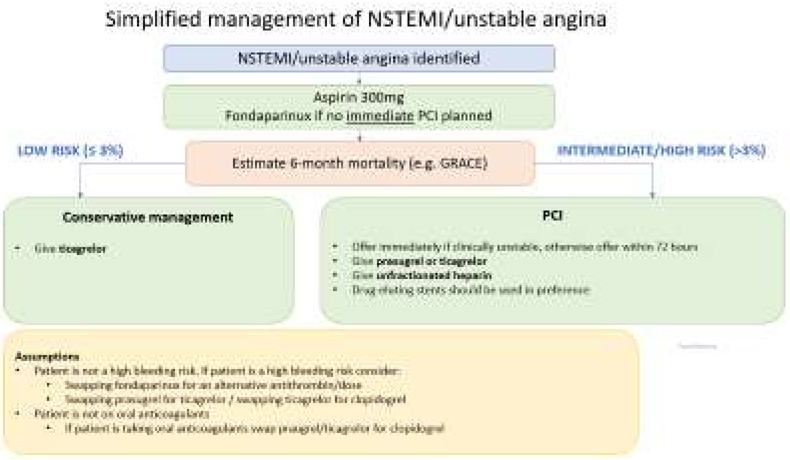

Simplified management of NSTEMI/unstable angina
NSTEMI/unstable angina identified
Aspirin 300mg
Fondaparinux if no immediate PCI planned
LOW RISK (≤ 3%)
Estimate 6-month mortality (e.g. GRACE)
INTERMEDIATE/HIGH RISK (>3%)
Conservative management
Give ticagrelor
PCI
Offer immediately if clinically unstable, otherwise offer within 72 hours
Give prasugrel or ticagrelor
Give unfractionated heparin
Drug eluting stents should be used in preference
Assumptions
Patient is not a high bleeding risk. If patient is a high bleeding risk consider:
Swapping fondaparinux for an alternative antithrombin/dose
Swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel/ticagrelor for clopidogrel

Diagram showing the simplified management of NSTEMI/unstable angina according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

The management of NSTEMI/unstable angina is complicated and depends on individual patient factors and a risk assessment. The summary below provides an overview but the full NICE guidelines should be reviewed for further details.

Further drug therapy
- antithrombin treatment
  - fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography immediately
  - if immediate angiography is planned or a patients creatinine is > 265 µmol/L then unfractionated heparin should be given

Risk assessment

The Global Registry of Acute Coronary Events (GRACE) is the most widely used tool for risk assessment. It can be calculated using online tools and takes into account the following factors:
- age
- heart rate, blood pressure
- cardiac (Killip class) and renal function (serum creatinine)
- cardiac arrest on presentation
- ECG findings
- troponin levels

This results in the patient being risk stratified as follows:

Predicted 6-month mortality	Risk of future adverse cardiovascular events
1.5% or below	Lowest
> 1.5% to 3.0%	Low
> 3.0% to 6.0%	Intermediate
> 6.0% to 9.0%	High
over 9.0%	Highest

Based on this risk assessment key decisions are made regarding whether a patient has coronary angiography (with follow-on PCI if necessary) or has conservative management. The detailed pros/cons of this decision are covered in other notes.

Which patients with NSTEMI/unstable angina should have coronary angiography (with follow-on PCI if necessary)?
- immediate: patient who are clinically unstable (e.g. hypotensive)
- within 72 hours: patients with a GRACE score > 3% i.e. those at intermediate, high or highest risk
- coronary angiography should also be considered for patients if ischaemia is subsequently experienced after admission

Percutaneous coronary intervention for patients with NSTEMI/unstable angina

Further drug therapy
- unfractionated heparin should be given regardless of whether the patient has had fondaparinux or not
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug) prior to PCI
  - if the patient is not taking an oral anticoagulant: prasugrel or ticagrelor
  - if taking an oral anticoagulant: clopidogrel

Conservative management for patients with NSTEMI/unstable angina

Further drug therapy

- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug)
    - if the patient is not at a high risk of bleeding: ticagrelor
    - if the patient is at a high risk of bleeding: clopidogrel

AI + Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 25 👎 28

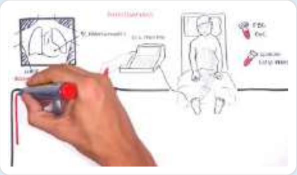
2016 Acute coronary syndrome guidelines

NICE 👍 64 👎 33

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Media



Acute Coronary Syndrome DETAILED Overview (MI, STEMI, NSTEMI)

Armando Hasudungan - YouTube 👍 5 👎 0

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 4 👎 1

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Score: **18.8%**

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You review a 65 year-old male who was admitted yesterday. He is a diabetic gentleman who was admitted with a non-ST elevation myocardial infarction and subsequent flash pulmonary oedema. He has been treated with aspirin, clopidogrel, fondaparinux and intravenous furosemide. He is achieving a good diuresis - producing 100mls of urine per hour.

You have been asked to see him as he continues to have significant breathlessness but no ongoing chest pain. His blood pressure is 92/87mmHg and oxygen saturations are 83% on 65% humidified oxygen. He has bibasal crepitations and JVP is raised. His ABG results are as follows:

pH	7.32
pCO2	4.6kPa
pO2	7.9kPa

What would be your next clinical intervention?

- ○ Start non-invasive ventilation
- ○ Start continuous positive airway pressure ventilation (CPAP)
- ○ Refer for angiography and primary coronary intervention
- ○ Start a tirofiban infusion
- ○ Start a GTN infusion

Submit answer

Reference ranges

You review a 65 year-old male who was admitted yesterday. He is a diabetic gentleman who was admitted with a non-ST elevation myocardial infarction and subsequent flash pulmonary oedema. He has been treated with aspirin, clopidogrel, fondaparinux and intravenous furosemide. He is achieving a good diuresis - producing 100mls of urine per hour.

You have been asked to see him as he continues to have significant breathlessness but no ongoing chest pain. His blood pressure is 92/87mmHg and oxygen saturations are 83% on 65% humidified oxygen. He has bibasal crepitations and JVP is raised. His ABG results are as follows:

pH	7.32
pCO2	4.6kPa
pO2	7.9kPa

What would be your next clinical intervention?

Start non-invasive ventilation	12%
Start continuous positive airway pressure ventilation (CPAP)	72%
Refer for angiography and primary coronary intervention	13%
Start a tirofiban infusion	1%
Start a GTN infusion	3%

Acute heart failure not responding to treatment - consider CPAP

Important for me Less important

This gentleman has had a non-ST elevation myocardial infarction (NSTEMI) and is now struggling with hypoxia secondary to pulmonary oedema. He has been treated optimally for his NSTEMI and is now pain free. He is diuresing well but continues to be hypoxic with signs of ongoing pulmonary oedema. The next logical intervention would be to try CPAP to improve his oxygenation. GTN, tirofiban and angiography would all look to improve coronary perfusion, however the gentleman is pain free suggesting this is not a priority.

Discuss (11) Improve

Next question

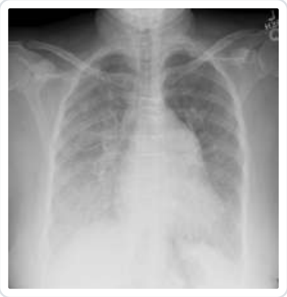
Heart failure: acute management

Recommended treatments for all patients

- IV loop diuretics
    - e.g. furosemide or bumetanide

Possible additional treatments

- oxygen
    - this should be given in line with British Thoracic Society guidelines, i.e. keep oxygen saturations at 94-98%
- vasodilators
    - nitrates should not be routinely given to all patients
    - they may, however, have a role if concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease
    - the major side-effect/contraindication is hypotension



Patients with respiratory failure

- CPAP

Patients with hypotension (e.g. < 85 mmHg)/cardiogenic shock

- this can be a difficult scenario to manage
    - some of the treatments typically used for acute heart failure (e.g. loop diuretics and nitrates) may exacerbate the hypotension
- inotropic agents
    - e.g. dobutamine
    - should be considered for patients with severe left ventricular dysfunction who have potentially reversible cardiogenic shock
- vasopressor agents
    - e.g. norepinephrine
    - normally only used if insufficient response to inotropes and evidence of end-organ hypoperfusion
- mechanical circulatory assistance
    - e.g. intra-aortic balloon counterpulsation or ventricular assist devices

Other treatments

- opiates
    - NICE state 'do not routinely offer opiates to people with acute heart failure'
    - they were previously used routinely and help to reduce dyspnoea/distress in patients
    - however some studies have suggested increased morbidity in patients given opiates

General points

- regular medication for heart failure such as beta-blockers ACE-inhibitors should be continued
    - beta-blockers should only be stopped if the patient has heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

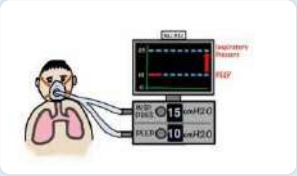
Links

NICE 👍 6 👎 9

2014 Acute heart failure: diagnosis and management

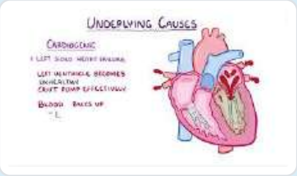
Suggest link Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 👍 17 👎 0



Pulmonary oedema

Osmosis - YouTube 👍 4 👎 0

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Question 157 of 328

A 72-year-old man attends cardiology clinic for routine follow-up of ischaemic heart disease. The patient had suffered a non-ST elevation myocardial infarction the previous year treated with a drug eluting stent (see below angiogram report). The patient reported being generally well over previous months although did experience retro-sternal chest pain on exertion, typically on walking up hills or climbing stairs. The patient denied any episodes of pain at rest and did not feel his symptoms were worsening. The patients regular medications were aspirin 75 mg daily, clopidogrel 75 mg daily, atorvastatin 80 mg daily and ramipril 5 mg daily. As required medications were sildenafil and a nitrate spray (which he reported as giving effective relief from his chest pain episodes).

Clinical examination did not demonstrate any evidence of cardiac failure. Blood pressure in clinic was 125 / 75 mmHg.

Results of investigations conducted 1 year previously following presentation with NSTEMI are included below.

Angiography: 90 % stenosis to left anterior descending artery treated with drug-eluting stent; 60 % stenosis to mid-right coronary artery; minor disease to other vessels.

Transthoracic echocardiogram: valvular function unremarkable; mild hypokinesis to anterior myocardium; overall ejection fraction approximately 40-45 %.

Electrocardiogram: sinus rhythm at 75 beats per minute; normal axis; normal QRS; no significant ST changes; inverted T waves in V3-V5.

What is most appropriate next line therapy for the patients chest pain?

- ○ Amlodipine ×
- ○ Bisoprolol ×
- ○ Percutaneous coronary intervention ×

○ Long-acting isosorbide mononitrate ×

○ Nicorandil ×

Submit answer

Reference ranges

Score: **18.7%**

1	✗
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A 72-year-old man attends cardiology clinic for routine follow-up of ischaemic heart disease. The patient had suffered a non-ST elevation myocardial infarction the previous year treated with a drug eluting stent (see below angiogram report). The patient reported being generally well over previous months although did experience retro-sternal chest pain on exertion, typically on walking up hills or climbing stairs. The patient denied any episodes of pain at rest and did not feel his symptoms were worsening. The patients regular medications were aspirin 75 mg daily, clopidogrel 75 mg daily, atorvastatin 80 mg daily and ramipril 5 mg daily. As required medications were sildenafil and a nitrate spray (which he reported as giving effective relief from his chest pain episodes).

Clinical examination did not demonstrate any evidence of cardiac failure. Blood pressure in clinic was 125 / 75 mmHg.

Results of investigations conducted 1 year previously following presentation with NSTEMI are included below.

Angiography: 90 % stenosis to left anterior descending artery treated with drug-eluting stent; 60 % stenosis to mid-right coronary artery; minor disease to other vessels.

Transthoracic echocardiogram: valvular function unremarkable; mild hypokinesis to anterior myocardium; overall ejection fraction approximately 40-45 %.

Electrocardiogram: sinus rhythm at 75 beats per minute; normal axis; normal QRS; no significant ST changes; inverted T waves in V3-V5.

What is most appropriate next line therapy for the patients chest pain?

Amlodipine	7%
Bisoprolol	68%
Percutaneous coronary intervention	14%

Long-acting isosorbide mononitrate	7%
Nicorandil	4%

The patients reported symptoms are consistent with a diagnosis of stable angina secondary to known ischaemic heart disease. Medical treatment for symptomatic relief is the appropriate next line therapy. In this patient with a history of NSTEMI and controlled blood pressure a beta-blocker is preferred over a calcium-channel blocker such as amlodipine.

Both ISMN and nicorandil are second-line options for medical therapy after intolerance or failure of beta-blocker and calcium-channel blocker therapy. Such both medications induce systemic vasodilatation they would be contraindicated in this patient while he still was taking sildenafil.

Revascularisation techniques such as PCI have not been show to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy should be attempted before consideration of invasive treatment.

Al-Lamee R, Davies J, Malik I. What is the role of coronary angioplasty and stunting in stable angina? BMJ 2016;352:i205.

👍 👎 Discuss (3) Improve

Next question >

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the

person's preference'

- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

Suggest link

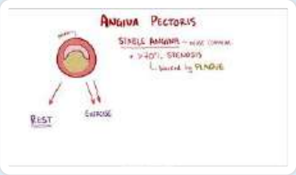
Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **19.2%**

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Question 158 of 328

A 74-year-old man presents to the emergency department with a collapse. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine and apixaban. A recent 24-hour tape arranged because of episodes of pre-syncope demonstrates sustained atrial fibrillation with episodes of bradycardia (minimum heart rate 20 beats per minute) associated with symptoms of pre-syncope.

The admission ECG demonstrates slow atrial fibrillation with a heart rate of 24 beats per minute.

The patient has recovered by the time he has been assessed by the medical team and his Glasgow coma scale is 15/15 and his heart rate is 82 beats per minute.

A decision is made to insert a permanent pacemaker.

What is the appropriate mode to programme the pacing system?

- ○ AAI
- ○ AOO
- ○ OOO
- ○ VAT
- ○ VVI

Submit answer

Reference ranges

Score: **19.2%**

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A 74-year-old man presents to the emergency department with a collapse. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine and apixaban. A recent 24-hour tape arranged because of episodes of pre-syncope demonstrates sustained atrial fibrillation with episodes of bradycardia (minimum heart rate 20 beats per minute) associated with symptoms of pre-syncope.

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The patient has recovered by the time he has been assessed by the medical team and his Glasgow coma scale is 15/15 and his heart rate is 82 beats per minute.

A decision is made to insert a permanent pacemaker.

What is the appropriate mode to programme the pacing system?

AAI	31%
AOO	4%
OOO	2%
VAT	8%
VVI	55%

VVI or VVR pacemakers are useful for pure sustained AF. Since the atria are not functioning they do not require sensing or pacing

Important for me Less important

VVI is the correct pacing mode. In this mode, the ventricle is sensed and the ventricle is paced. If a QRS complex is detected, it doesn't fire. If it is not detected i.e. there has been no ventricular electrical activity within the interval specified by its set rate, then it will fire. This is suitable for atrial fibrillation as the atria are not working correctly and there will be no detectable P wave and therefore the atria do not need to be sensed or paced.

AAI is not the correct pacing mode to choose. AAI pacing mode means atrial paced, atrial sensed, and inhibition of pacing output in response to an atrial sensed event (P wave). Since the atria are not functioning in AF, they do not require sensing or pacing. AAI pacing has better hemodynamic features than dual-chamber pacing and is the best mode for patients with sick sinus syndrome without a significant AV conduction disorder. This mode essentially relies on the AV node to fire and guarantees a certain atrial rate.

AOO is incorrect. This is asynchronous pacing. This refers to the continuous pacing of the atria irrespective of the presence or absence of an intrinsic rhythm. This pacing mode is limited to rare cases, such as when pacemaker-dependent patients (who do not have a ventricular paced setting) are subject to artefact (e.g., electrocautery during surgery), which could result in asystole due to oversensing and pacing inhibition if the pacemaker has been set to a non-asynchronous mode. It is never a permanent mode.

OOO is incorrect. This essentially means there is no sensing, no pacing and no response to sensing. It means the pacemaker is turned off.

VAT is incorrect. This mode means the atria are sensed and the ventricles are paced. This is suitable for a patient with a dysfunctional AV node but working atria. For example, in someone with a complete heart block and a normal atrial rate, a VAT pacemaker will sense the atrium and pace the ventricle in response.

👍 👎 Discuss (4) Improve

Next question >

Pacemaker: permanent ★

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:
- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):
- V - Ventricle

- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):

- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):

- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):

- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:

- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:

- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

DUAL CHAMBER
PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **19.7%**

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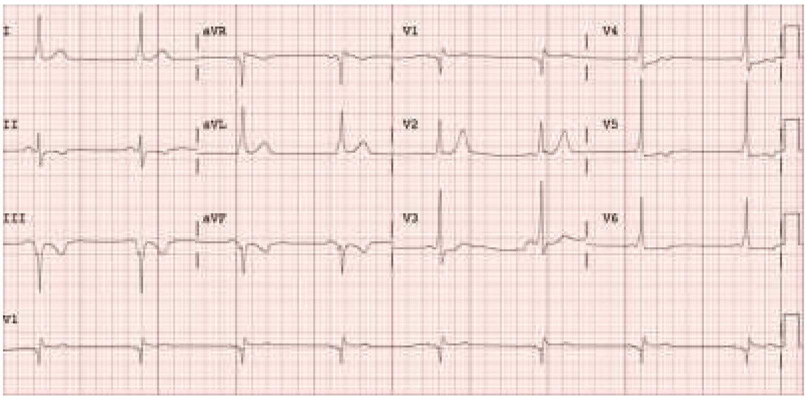
Question 159 of 328

A 35-year-old man is investigated for recurrent palpitations associated with pre-syncopal symptoms and dyspnoea. Blood tests are unremarkable and a resting 12 lead ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

Given the likely diagnosis it is decided that radiofrequency ablation is the most appropriate treatment. What part of the heart should be ablated?

○ Sinoatrial node

○ Atrioventricular node

○ Junction of right atrium and right ventricle

○ Left atrial appendage

○ Junction of left atrium and left ventricle

Submit answer

Reference ranges

Score: **19.7%**

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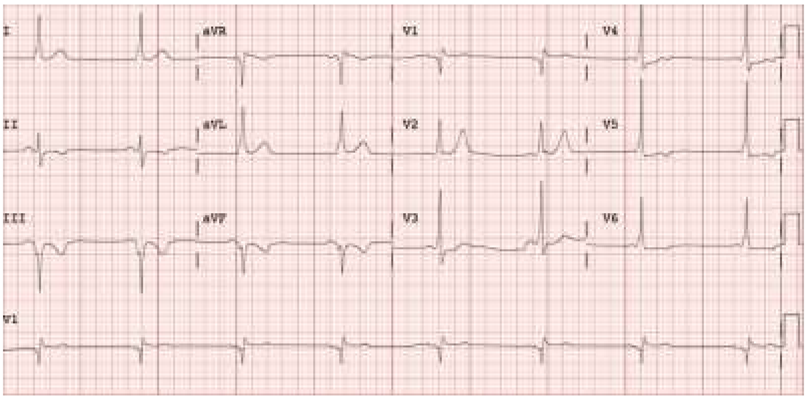
Question 159 of 328 ✗

A 35-year-old man is investigated for recurrent palpitations associated with pre-syncopal symptoms and dyspnoea. Blood tests are unremarkable and a resting 12 lead ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

Given the likely diagnosis it is decided that radiofrequency ablation is the most appropriate treatment. What part of the heart should be ablated?

	Sinoatrial node	5%
	Atrioventricular node	12%
🟩	Junction of right atrium and right ventricle	49%
	Left atrial appendage	14%
🟥	Junction of left atrium and left ventricle	21%

The ECG shows a short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway.

Discuss (6) | Improve

Next question >

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

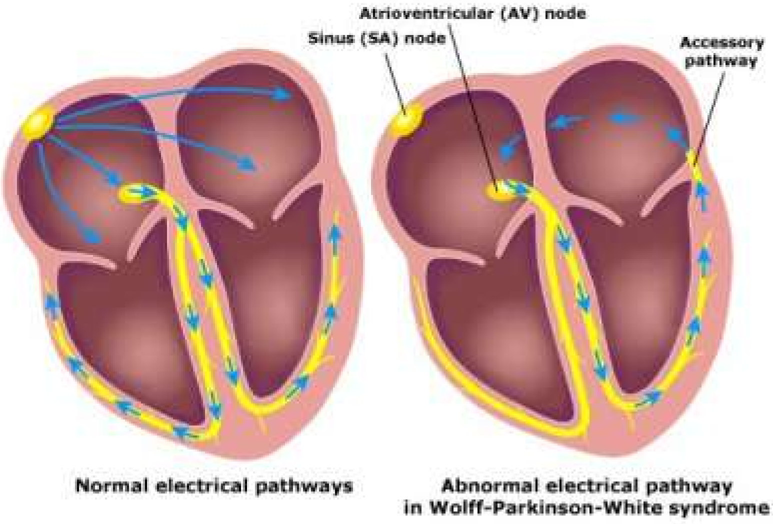
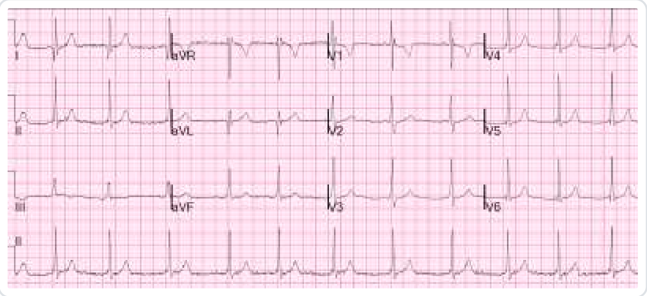

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
    - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway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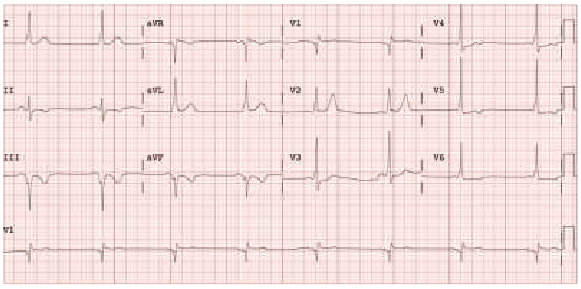

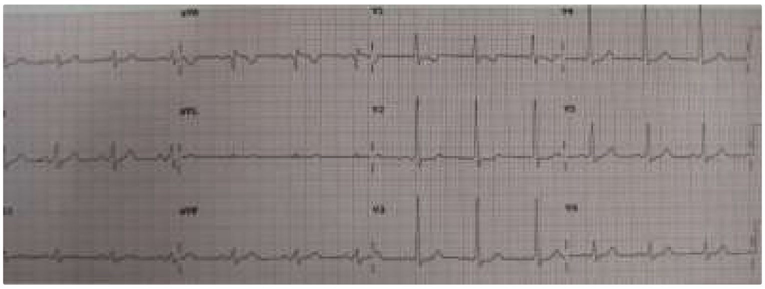

Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
    - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 👍 12 👎 2

Report broken media

Score: **19.6%**

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157 ✓

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159 ✗

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A 34-year-old male presents to the emergency department with chest tightness worse on inspiration. He informs you that over the past 3 days he has had a low-grade temperature and a degree of myalgia. Prior to all this he had no limitations to his activity and never experienced any chest tightness. He was given glyceryl trinitrate spray and aspirin in the ambulance, neither of which have made any difference.

Examination is unremarkable, however you note that his temperature is 37.4ºC. He is a smoker and he informs you that his father, mother and paternal uncle all have had heart attacks around the age of 60. His bloods are the following:

Hb	165 g/L	Male: (135-180) Female: (115 - 160)
Platelets	537 * 10^9/L	(150 - 400)
WBC	12.3 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Urea	4.6 mmol/L	(2.0 - 7.0)
Creatinine	93 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)
Troponin T (0hrs)	586 ng/L	(<14)
Troponin T (3hrs)	615 ng/L	(<14)
D-dimer	Negative	

An ECG is performed indicating sinus rhythm, no intraventricular delay and widespread ST elevations; you also notice was subtle PR depression.

What treatment should be initiated in the case of this gentleman?

○ Arrange urgent angioplasty and potential revascularisation

○ Load patient with aspirin and clopidogrel

○ Load patient with aspirin and ticagrelor ×

○ Prescribe ibuprofen TDS ×

○ Prescribe ibuprofen TDS and colchicine BD ×

Submit answer

Reference ranges ∨

Score: **19.6%**

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Back to top

Question 160 of 328 ✓

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D-dimer	Negative	

An ECG is performed indicating sinus rhythm, no intraventricular delay and widespread ST elevations; you also notice was subtle PR depression.

What treatment should be initiated in the case of this gentleman?

- Arrange urgent angioplasty and potential revascularisation 3%
- Load patient with aspirin and clopidogrel 1%

Load patient with aspirin and ticagrelor	1%
Prescribe ibuprofen TDS	21%
Prescribe ibuprofen TDS and colchicine BD	75%

First line management of acute pericarditis involves combination of NSAID and colchicine

Important for me Less important

The history, examination and blood investigations are consistent with acute pericarditis/myopericarditis. The high troponin results indicate myocardial injury which in this case can be attributed to myopericarditis. Troponin is a marker of myocardial injury which can result due to primary ischaemia (plaque rupture or intraluminal coronary artery thrombus), ischaemia secondary to misbalance of supply and demand (tachy/bradyarrhythmias, aortic dissection or severe aortic valve disease, sepsis, severe anaemia or respiratory failure etc.) and myocardial injury not necessarily associated to ischaemia or multifactorial (such as myopericarditis, severe pulmonary embolism with right heart strain, cardiac stunning, cardiac surgery or intervention etc.) There is no strong, dynamic rise in the already high troponin level.

The fact that he is a smoker undeniably puts this gentleman at a higher risk of coronary event, however his blood tests, ECG and history point further to the direction of pericarditis. Given this, options 1, 2 and 3 which are associated with the management of acute coronary syndrome (ACS) are wrong.

The current guidelines as per the European Society of cardiology state that the treatment of choice in such patients is a combination of non-steroidal anti-inflammatory drugs (e.g. ibuprofen) and colchicine (option 5) and do not advocate the use of a single agent, such as ibuprofen (option 4).

👍 👎 Discuss (3) Improve

Next question ›

Acute pericarditis ★

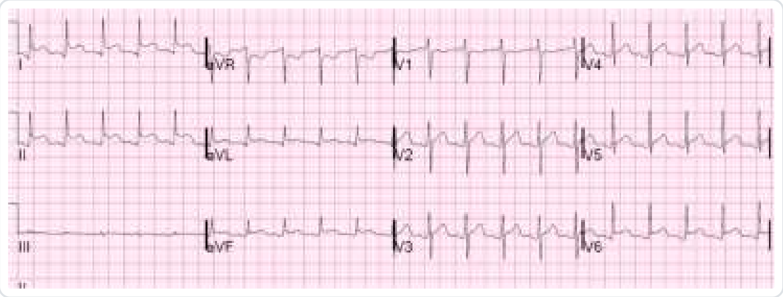
Acute pericarditis is a condition referring to inflammation of the pericardial sac, lasting for less than 4-6 weeks.

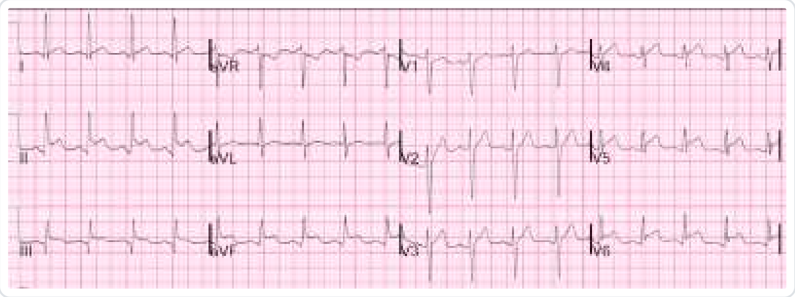
Aetiology
- viral infections (Coxsackie)
- tuberculosis
- uraemia
- post-myocardial infarction
  - early (1-3 days): fibrinous pericarditis
  - late (weeks to months): autoimmune pericarditis (Dressler's syndrome)
- radiotherapy
- connective tissue disease
  - systemic lupus erythematosus
  - rheumatoid arthritis
- hypothyroidism
- malignancy
  - lung cancer
  - breast cancer
- trauma

Features
- chest pain: may be pleuritic. Is often relieved by sitting forwards
- other symptoms include a non-productive cough, dyspnoea and flu-like symptoms
- pericardial rub

Investigations
- ECG changes
  - the changes in pericarditis are often global/widespread, as opposed to the 'territories' seen in ischaemic events
  - 'saddle-shaped' ST elevation
  - PR depression: most specific ECG marker for pericarditis
- all patients with suspected acute pericarditis should have transthoracic echocardiography
- bloods
  - inflammatory markers

    - troponin: around 30% of patients may have an elevated troponin - this indicates possible myopericarditis





Management

- the majority of patients can be managed as outpatients
    - patients who have high-risk features such as fever > 38°C or elevated troponin should be managed as an inpatient
- treat any underlying cause
    - most patients however will have pericarditis secondary to viral infection, meaning no specific treatment is indicated
- strenuous physical activity should be avoided until symptom resolution and normalisation of inflammatory markers
- a combination of NSAIDs and colchicine is now generally used for first-line for patients with acute idiopathic or viral pericarditis
    - until symptom resolution and normalisation of inflammatory markers (usually 1-2 weeks) followed by tapering of dose recommended over

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 35 👎 15

Pericarditis review

Suggest link

Report broken link

Media

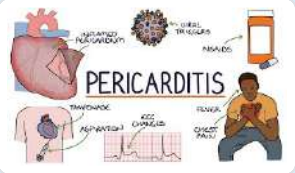
Pericardial rub - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 0

PERICARDITIS &
PERICARDIAL
EFFUSIONS
Osmosis.org

Pericarditis and pericardial effusions

Osmosis - YouTube 👍 2 👎 0


PERICARDITIS

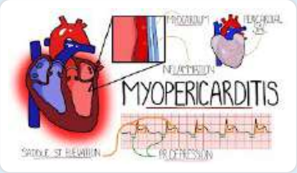
Pericarditis

Zero To Finals - YouTube 👍 0 👎 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 👍 2 👎 1


MYOPERICARDITIS
INFLAMMATION
MYOCARDIUM
PERICARDIAL
SADDLE ST ELEVATION
PR DEPRESSION

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **20.1%**

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160	✓

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A 55-year-old woman presents to the emergency department with chest pain. She is currently pain free, but has had two similar episodes recently lasting approximately 2-3 minutes. She describes the pain as squeezing and central and feels that the pain is brought on by exertion and relieved within a few minutes by resting. Her father died of a heart attack at 72, she has never smoked and has no significant medical history.

On examination, she is slim, her blood pressure is 125/75 mmHg and heart rate is 85 beats per minute, no murmurs are heard and chest sounds are normal. A 12-lead ECG shows normal sinus rhythm.

serum cholesterol	3.2 mmol/L (<5.2)
serum LDL cholesterol	2.0 mmol/L (<3.36)
serum HDL cholesterol	2.65 mmol/L (>1.55)
fasting serum triglycerides	1.0 mmol/L (0.45-1.69)
haemoglobin	125 g/L (115-165)

What is the next most appropriate investigation?

- ○ Invasive coronary angiography
- ○ CT coronary angiography
- ○ Exercise tolerance test
- ○ Myocardial perfusion scintigraphy with single photon emission computed tomography
- ○ Stress echocardiogram

Submit answer

Reference ranges

A 55-year-old woman presents to the emergency department with chest pain. She is currently pain free, but has had two similar episodes recently lasting approximately 2-3 minutes. She describes the pain as squeezing and central and feels that the pain is brought on by exertion and relieved within a few minutes by resting. Her father died of a heart attack at 72, she has never smoked and has no significant medical history.

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fasting serum triglycerides	1.0 mmol/L (0.45-1.69)
haemoglobin	125 g/L (115-165)

What is the next most appropriate investigation?

Invasive coronary angiography	2%
CT coronary angiography	63%
Exercise tolerance test	21%
Myocardial perfusion scintigraphy with single photon emission computed tomography	6%
Stress echocardiogram	7%

Discuss (6) Improve

Chest pain: assessment of patients with suspected cardiac chest pain ★

NICE updated it's guidelines in 2016 on the 'Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin'.

Below is a brief summary of the key points. Please see the link for more details.

Patients presenting with acute chest pain

Immediate management of suspected acute coronary syndrome (ACS)
- glyceryl trinitrate
- aspirin 300mg. NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital
- do not routinely give oxygen, only give if sats < 94%*
- perform an ECG as soon as possible but do not delay transfer to hospital. A normal ECG does not exclude ACS

Referral
- current chest pain or chest pain in the last 12 hours with an abnormal ECG: emergency admission
- chest pain 12-72 hours ago: refer to hospital the same-day for assessment
- chest pain > 72 hours ago: perform full assessment with ECG and troponin measurement before deciding upon further action

*NICE suggest the following in terms of oxygen therapy:
- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is available.

Patients presenting with stable chest pain

NICE define anginal pain as the following:

- 1. constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arms
- 2. precipitated by physical exertion
- 3. relieved by rest or GTN in about 5 minutes
- patients with all 3 features have typical angina
- patients with 2 of the above features have atypical angina
- patients with 1 or none of the above features have non-anginal chest pain

For patients in whom stable angina cannot be excluded by clinical assessment alone NICE recommend the following (e.g. symptoms consistent with typical/atypical angina OR ECG changes):

- 1st line: CT coronary angiography
- 2nd line: non-invasive functional imaging (looking for reversible myocardial ischaemia)
- 3rd line: invasive coronary angiography

Examples of non-invasive functional imaging:

- myocardial perfusion scintigraphy with single photon emission computed tomography (MPS with SPECT) or
- stress echocardiography or
- first-pass contrast-enhanced magnetic resonance (MR) perfusion or
- MR imaging for stress-induced wall motion abnormalities

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 14 👎 7

2013 ESC guidelines on the management of stable coronary artery disease

NICE 👍 11 👎 3

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Suggest link Report broken link

Score: **20%**

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161 ✗

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Question 162 of 328

A 65-year-old man presents to his general practitioner with exertional chest tightness. This occurs stereotypically after walking approximately 100 meters. It stops when he rests. He has a past medical history of hypertension and asthma. His medications include aspirin, atorvastatin, amlodipine, and a regular inhaler.

The examination is unremarkable. His blood pressure is 154/42 mmHg.

What is the most appropriate choice of medication to control his symptoms?

○ Atenolol ×
○ Bisoprolol ×
○ Diltiazem ×
○ Ramipril ×
○ Ranolazine ×

Submit answer

Reference ranges

Score: **20%**

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162	-

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Question 162 of 328 ✓

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The examination is unremarkable. His blood pressure is 154/42 mmHg.

What is the most appropriate choice of medication to control his symptoms?

Atenolol	5%
Bisoprolol	25%
Diltiazem	24%
Ramipril	14%
Ranolazine ✓	32%

For a patient with symptomatic stable angina on a calcium channel blocker but with a contraindication to a beta-blocker, the next line treatment should be long-acting nitrate, ivabradine, nicorandil or ranolazine

Important for me   Less important

Ranolazine is correct. The patient has symptomatic stable angina. A beta blocker would be an obvious choice to add at this point but is contraindicated in this patient due to their history of asthma. It may precipitate bronchospasm due to the effect on beta-adrenoreceptors. The mechanism of action of ranolazine is largely unknown. Ranolazine may have antianginal effects by inhibition of the late sodium current in cardiac myocytes.

Atenolol is incorrect. Beta-blockers are contraindicated in this patient due to the history of asthma.

Bisoprolol is incorrect. This is another beta-blocker. It is more cardioselective than atenolol but still would not be a prudent choice for a patient with asthma when there are alternatives available.

Diltiazem is incorrect. This is another calcium-channel blocker. It should not be used together with amlodipine, which is also a calcium channel blocker. Diltiazem is a non-dihydropyridine calcium channel blocker while amlodipine is a dihydropyridine calcium channel blocker. Dihydropyridine calcium channel antagonists cause predominantly peripheral vasodilatory actions. Nondihydropyridine CCBs have sinoatrial (SA) and AV node depressant effects with lesser amounts of peripheral vasodilation.

Ramipril is incorrect. This has predominantly anti-hypertensive rather than anti-anginal effects. The blood pressure in this patient is elevated but remains at stage 1 grade. Additionally, we were asked to choose a medication to help his *symptoms* rather than control his blood pressure.

👍 👎 💬 Discuss (6) Improve

Next question ›

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication
- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-

release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 21 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

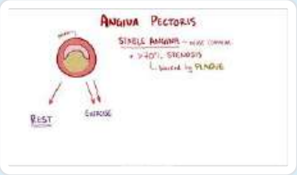
Suggest link Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
>70% STENOSIS
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **20.5%**

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A 16-year-old female presents with a swollen knee. She states that she has had several similar episodes previously. She also complains of excessive bleeding after dental work. She states that her father has a bleeding disorder. On examination her right knee is hot and swollen. A knee aspirate reveals a bloody effusion.

Blood results are as follows:

Hb	110 g/l
Platelets	682 * 10^9/l
Prothrombin time (PT)	12 s (normal 10-13)
activated partial thromboplastin time (aPTT)	54 s (normal 25-36)
Factor VIII level	2% of normal (very low)
Factor IX level	Normal
von Willebrand Factor level	Normal

What disorder does she most likely have?

- ○ Down syndrome ×
- ○ Edwards syndrome ×
- ○ Marfan syndrome ×
- ○ Ehlers-Danlos syndrome ×
- ○ Turner's syndrome ×

Submit answer

Reference ranges

A 16-year-old female presents with a swollen knee. She states that she has had several similar episodes previously. She also complains of excessive bleeding after dental work. She states that her father has a bleeding disorder. On examination her right knee is hot and swollen. A knee aspirate reveals a bloody effusion.

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activated partial thromboplastin time (aPTT)	54 s (normal 25-36)
Factor VIII level	2% of normal (very low)
Factor IX level	Normal
von Willebrand Factor level	Normal

What disorder does she most likely have?

Down syndrome	5%
Edwards syndrome	20%
Marfan syndrome	7%
Ehlers-Danlos syndrome	29%
Turner's syndrome	38%

Females with Turner's syndrome have just one X chromosome. Therefore they have the same probability of being affected by an X linked recessive disease as males

Important for me Less important

The patient clearly has haemophilia A as indicated by the prolonged aPTT and severely reduced factor VIII levels. Rare cases of females with severe haemophilia can occur due to extreme lyonization, homozygosity, mosaicism, or Turner syndrome.

Females with Turner's syndrome have just one X chromosome. Thus they have the same probability of being affected by an X linked recessive disease as males. For the aforementioned reason, Turner's syndrome is the most likely underlying diagnosis out of the given options.

👍 👎 Discuss (4) Improve

Next question >

Turner's syndrome ★

Turner's syndrome is a chromosomal disorder affecting around 1 in 2,500 females. It is caused by either the presence of only one sex chromosome (X) or a deletion of the short arm of one of the X chromosomes. Turner's syndrome is denoted as 45,XO or 45,X.

Features
- short stature
- shield chest, widely spaced nipples
- webbed neck
- bicuspid aortic valve (15%), coarctation of the aorta (5-10%)
  - an increased risk of aortic dilatation and dissection are the most serious long-term health problems for women with Turner's syndrome
  - regular monitoring in adult life for these complications is an important component of care
- primary amenorrhoea
- cystic hygroma (often diagnosed prenatally)
- high-arched palate
- short fourth metacarpal
- multiple pigmented naevi
- lymphoedema in neonates (especially feet)
- gonadotrophin levels will be elevated
- hypothyroidism is much more common in Turner's
- horseshoe kidney: the most common renal abnormality in Turner's syndrome

There is also an increased incidence of autoimmune disease (especially autoimmune thyroiditis) and Crohn's disease

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

TURNER
SYNDROME
Osmosis.org

Turner's syndrome

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **20.4%**

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Question 164 of 328

A 68-year-old man is referred to the cardiology outpatient department with shortness of breath on exertion. He becomes breathless when ascending stairs. There is no chest pain. He has a past medical history of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 83 beats per minute, blood pressure 110/85 mmHg, respiratory rate 18/minute, oxygen saturations 97% on room air and temperature 37.1°C.

On examination, he is euvolemic. His heart sounds are normal and there is no peripheral oedema. Chest auscultation is normal. The pulse is regular.

From the options listed, what is the best choice of medication to improve his symptoms?

- ○ Flecainide
- ○ Ivabradine
- ○ Nicorandil
- ○ Ranolazine
- ○ Verapamil

Submit answer

Reference ranges

Score: **20.4%**

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A 68-year-old man is referred to the cardiology outpatient department with shortness of breath on exertion. He becomes breathless when ascending stairs. There is no chest pain. He has a past medical history of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 83 beats per minute, blood pressure 110/85 mmHg, respiratory rate 18/minute, oxygen saturations 97% on room air and temperature 37.1ºC.

On examination, he is euvolemic. His heart sounds are normal and there is no peripheral oedema. Chest auscultation is normal. The pulse is regular.

From the options listed, what is the best choice of medication to improve his symptoms?

Flecainide	1%
Ivabradine	87%
Nicorandil	5%
Ranolazine	5%
Verapamil	3%

Ivabradine should be considered in heart failure if the patient has sinus rhythm > 75/min and a LVEF < 35% and have not responded to to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Important for me Less important

Ivabradine is the correct answer. This man has NYHA class II/III heart failure despite treatment with an ace inhibitor, beta-blocker and mineralocorticoid receptor antagonist. Ivabradine is an option in heart failure for those patients who have not responded to the previously described combination of medications and have a heart rate > 75 beats per minute and an LVEF < 35%.

Nicorandil and ranolazine are incorrect. These medications are options for the treatment of angina, not heart failure.

Flecainide is incorrect. This is a medication used to chemically cardiovert atrial fibrillation. This patient is in sinus rhythm.

Verapamil is incorrect. Negatively inotropic calcium channel blockers should be avoided in heart failure, especially in the context of current beta blocker treatment.

👍 👎 Discuss (1) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**
- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

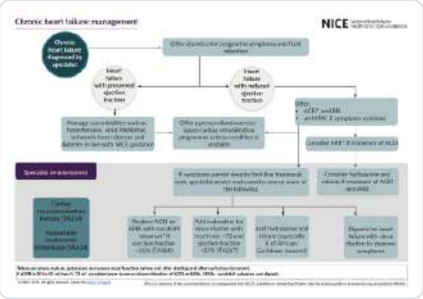
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy

    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years



123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

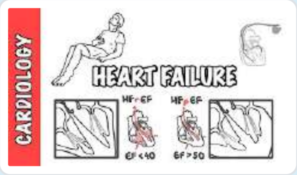
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
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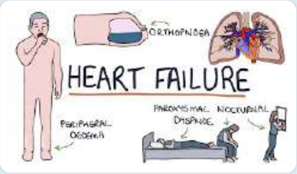
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


HEART FAILURE
ORTHOPNOEA
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **20.2%**

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Back to top

A 102-year-old man with a previous history of heart failure, amyloid disease and a permanent pacemaker attends the emergency department due to a syncopal episode. He recalls passing out in his chair whilst watching TV and was attended to by nursing home staff who were able to rouse him after 2 minutes of loss of consciousness. This is the third episode in the past week.

On examination, his heart rate is 32 beats per minute and irregular. He has crepitations in his chest and a raised JVP but no leg oedema. He is alert but cool peripherally. He has no pain at present.

Na^+	135 mmol/l
K^+	4.5 mmol/l
Urea	5.6 mmol/l
Creatinine	76 µmol/l
ECG	p-waves present with no proceeding QRS, widened QRS complexes seen
Chest x-ray	chronic lower zone changes, pacemaker leads have a necked appearance

What is the likely diagnosis?

- ○ Lead fracture
- ○ Lead displacement
- ○ Twiddlers syndrome
- ○ Silent myocardial infarction
- ○ Progression of cardiomyopathy

Submit answer

Reference ranges ﹀

Score: **20.2%**

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Question 165 of 328 ✗

A 102-year-old man with a previous history of heart failure, amyloid disease and a permanent pacemaker attends the emergency department due to a syncopal episode. He recalls passing out in his chair whilst watching TV and was attended to by nursing home staff who were able to rouse him after 2 minutes of loss of consciousness. This is the third episode in the past week.

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Na^+	135 mmol/l
K^+	4.5 mmol/l
Urea	5.6 mmol/l
Creatinine	76 µmol/l
ECG	p-waves present with no proceeding QRS, widened QRS complexes seen
Chest x-ray	chronic lower zone changes, pacemaker leads have a necked appearance

What is the likely diagnosis?

Lead fracture	40%
Lead displacement	31%
Twiddlers syndrome	21%
Silent myocardial infarction	2%
Progression of cardiomyopathy	6%

Early signs of pacemaker lead failure are a 'necked' appeared on imaging

Important for me Less important

This gentleman has complete heart block despite a permanent pacemaker in situ. Cardiomyopathy progression and silent myocardial infarction would be detected by progression of congestive cardiac failure with worsening pulmonary or peripheral oedema. The presence of complete heart block suggests that the pacemaker may not be functioning correctly. It is noted that the leads have a necked appearance and this occurs after repeat bending of the leads that causes them to weaken and eventually fracture. This is a rare complication.

Despite being intact on chest x-ray. the necking suggests that the wires are starting to fray and function can be lost prior to a complete fracture of the lead is seen. Twiddling of the wires or lead displacement would be seen on chest x-ray.

Discuss (10) Improve

Next question

Pacemaker: permanent

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:
- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no pacing)

Second Position (Chamber Sensed):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):
- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):

- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):

- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:

- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:

- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.
- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

AI + Q 123

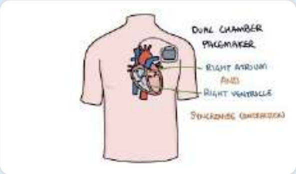
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


DUAL CHAMBER PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE
SYNCHRONISE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **20.1%**

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Question 166 of 328

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (28%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 58/min with a QRS of 98 msec and no signs of intraventricular delay. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	129 g/L	Male: (135-180) Female: (115 - 160)
Platelets	335 * 10^9/L	(150 - 400)
WBC	6.8* 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	125 µmol/L	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD, spironolactone 25 mg OD.

What alteration to this gentleman's medications could potentially decrease this gentleman's probability of being re-admitted?

- ○ Increase ivabradine ×
- ○ Add sacubitril/valsartan ×
- ○ Increase spironolactone ×
- ○ Stop enalapril - initiate sacubitril/valsartan ×
- ○ Increase bumetanide ×

Submit answer

Reference ranges ⌄

Score: **20.1%**

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Back to top

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (28%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 58/min with a QRS of 98 msec and no signs of intraventricular delay. His blood pressure is: 98/55 mmHg. You also review his bloods:

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He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD, spironolactone 25 mg OD.

What alteration to this gentleman's medications could potentially decrease this gentleman's probability of being re-admitted?

Increase ivabradine	2%
Add sacubitril/valsartan	8%
Increase spironolactone	4%
Stop enalapril - initiate sacubitril/valsartan	85%
Increase bumetanide	1%

Sacubitril-valsartan should be initiated following ACEi or ARB wash-out period

Important for me Less important

This gentleman poses the issue of long-standing management of heart-failure. The ESC guidelines advice strictly on the uptitration of medication up to their maximal desired effect taking in consideration the patient's status - the frontline medications include ACE inhibitors, b-blockers and mineralocorticoid receptor antagonists. The patient is on all three. If the patient remains symptomatic then a good choice would be dependable on their QRS morphology with a CRT-P device being appropriate in certain cases (e.g. QRS >130 msec) or ivabradine in someone with a heart rate of more than 70/min. The option of digoxin is also discussed in the ESC guidelines.

However the patient above is already bradycardic and hypotensive with a slightly raised potassium therefore making the possibility of increasing any of his current medications less appealing. In these patients the possibility of introducing sacubitril/valsartan is appropriate, however it is important for the clinician to be aware of the need for a wash-out period between the ACEi and the initiation of sacubitril/valsartan. The wash-out period advised is that of 36 hours.

The increase in diuretic might appear appealing, there is no evidence that diuretics improve mortality rates in such patients. The PARADIGM-HF trial that was comparing sacubitril/valsartan and enalapril indicated that sacubitril/valsartan was superior to enalapril in reducing cardiovascular mortality, heart failure hospitalisation and 30-day hospital readmission in heart failure patients with reduced ejection fraction.

👍 👎 Discuss (6) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

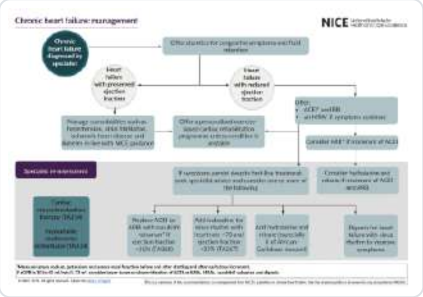
Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine

    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 12

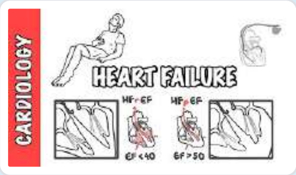
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

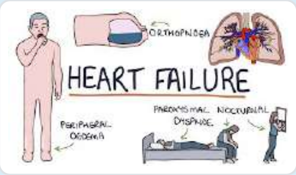
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1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HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **20%**

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Question 167 of 328

An 80-year-old man with a history of atrial fibrillation is admitted following an acute coronary syndrome and is treated with percutaneous coronary intervention. His medication on admission was warfarin, co-codamol and allopurinol.

With regards to antithrombotic therapy, what should he be prescribed in the immediate aftermath of the event?

- ○ Continue warfarin with the addition of 2 antiplatelets ×
- ○ Continue warfarin with the addition of 1 antiplatelet ×
- ○ Continue warfarin monotherapy ×
- ○ Stop warfarin, start 2 antiplatelets ×
- ○ Stop warfarin, start 3 antiplatelets ×

Submit answer

Reference ranges

Score: **20%**

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Question 167 of 328 ✕

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With regards to antithrombotic therapy, what should he be prescribed in the immediate aftermath of the event?

Continue warfarin with the addition of 2 antiplatelets	45%
Continue warfarin with the addition of 1 antiplatelet	35%
Continue warfarin monotherapy	5%
Stop warfarin, start 2 antiplatelets	13%
Stop warfarin, start 3 antiplatelets	1%

In the initial phase following an ACS/PCI patients who also have AF are generally given 2 antiplatelets + 1 anticoagulant

Important for me   Less important

Discuss (5)   Improve

Next question ›

Combination antiplatelet and anticoagulant therapy ★

With the increase in comorbidity, it is now common to find that a patient has an indication for both an antiplatelet (e.g. established cardiovascular disease) and an anticoagulant (e.g. atrial fibrillation, venous thromboembolism or valvular heart disease). However, combination therapy increases the risk of bleeding and may not be needed in all cases. How should this be managed?

Whilst there are not guidelines to cover every scenario a recent review in the BMJ offered an expert opinion outlining the approach in common scenarios.

Secondary prevention of stable cardiovascular disease with an indication for an anticoagulant
- normally in this situation, all patients are recommended to be prescribed an antiplatelet
- if an indication for anticoagulant exists (for example atrial fibrillation) it is indicated that anticoagulant monotherapy is given without the addition of antiplatelets

Post-acute coronary syndrome/percutaneous coronary intervention
- in these patients, there is a much stronger indication for antiplatelet therapy
- generally patients are given triple therapy (2 antiplatelets + 1 anticoagulant) for 4 weeks-6 months after the event and dual therapy (1 antiplatelet + 1 anticoagulant) to complete 12 months
- there is variation from patient to patient however given that the stroke risk in atrial fibrillation varies according to risk factors.

Venous thromboembolism (VTE)
- if a patient on antiplatelets develops a VTE they are likely to be prescribed anticoagulants for 3-6 months
- an ORBIT score should be calculated. Those with a low risk of bleeding may continue antiplatelets. In patients with an intermediate or high risk of bleeding consideration should be given to stopping the antiplatelets

For more information please see BMJ 2017;358:j3782

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

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Question 168 of 328

You are working in the general medical clinic where a 42 year old woman comes for review following a recent, short admission to hospital where she was treated for a paracetamol overdose. She has a past history of depression but denies any other previous problems.

During the review, she is found to have a manual blood pressure reading of 165/85 mmHg. Clinical examination of cardiovascular and respiratory systems are normal, as is urine dip and fundoscopy. Given this information what should be your next course of management in relation to her blood pressure.

- ○ Start ramipril
- ○ Offer ambulatory blood pressure monitoring
- ○ Arrange to check blood pressure again following a two week interval
- ○ Start amlodipine
- ○ Screen for causes of secondary hypertension

Submit answer

Reference ranges

Score: **19.9%**

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You are working in the general medical clinic where a 42 year old woman comes for review following a recent, short admission to hospital where she was treated for a paracetamol overdose. She has a past history of depression but denies any other previous problems.

During the review, she is found to have a manual blood pressure reading of 165/85 mmHg. Clinical examination of cardiovascular and respiratory systems are normal, as is urine dip and fundoscopy. Given this information what should be your next course of management in relation to her blood pressure.

Start ramipril	10%
Offer ambulatory blood pressure monitoring	77%
Arrange to check blood pressure again following a two week interval	7%
Start amlodipine	2%
Screen for causes of secondary hypertension	4%

In 2011 the National Institute for Clinical Excellence updated its 2006 guideline for the management of hypertension (see the link below for the quick reference guide). Within this guideline, the first line use of ambulatory blood pressure monitoring (ABPM) to confirm hypertension in those found to have an elevated clinic reading (> 140/90 mmHg) is emphasised. When using ABPM to confirm a diagnosis of hypertension, two measurements per hour are taken during the persons waking hours. The average value of at least 14 measurements are then used to confirm a diagnosis of hypertension.

Generally speaking, secondary causes of hypertension should be sought in; patients under 40 who lack traditional risk factors for essential hypertension, patients with other sings and/or symptoms of secondary causes, and patients with resistant hypertension. Although in reality the most common cause of secondary hypertension is hyperaldosteroneism, and as such a trial of an aldosterone antagonist such as spironolactone is often employed as both a therapeutic and diagnostic measure.

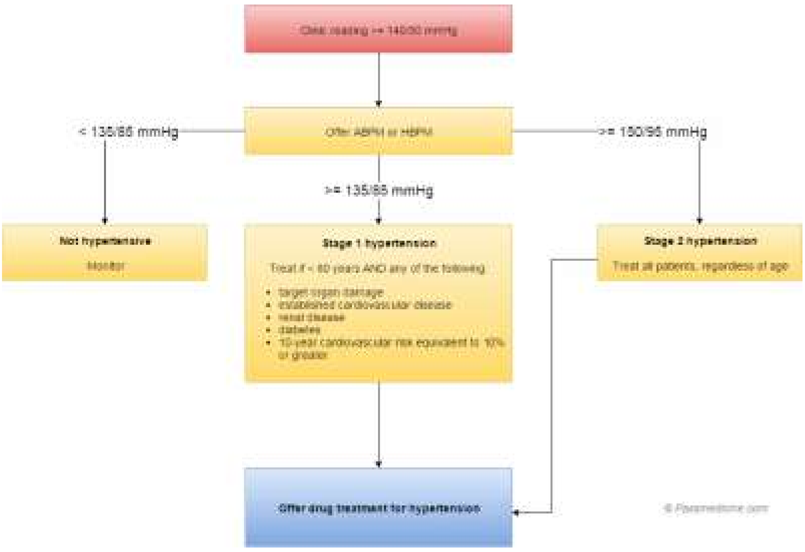
Drug treatment of essential hypertension can be summarised as follows, but for a more detailed explanation see the link below;
- Step 1; Age <55 - ACE inhibitor. Age >55 or of black African or Caribbean origin - calcium channel blocker
- Step 2; ACE inhibitor + calcium channel blocker
- Step 3; ACE inhibitor + calcium channel blocker + thiazide-like diuretic
- Step 4; consider further diuretic or beta-blockade or alpha blocker and seeking expert advice

Discuss (4) Improve

Next question >

Hypertension: diagnosis ★

NICE published updated guidelines for the management of hypertension in 2019. This builds on the significant guidelines released in 2011 that recommended:
- classifying hypertension into stages
- recommending the use of ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM)


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 135/85 mmHg
>= 150/95 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Why were these guidelines needed?

It has long been recognised by doctors that there is a subgroup of patients whose blood pressure climbs 20 mmHg whenever they enter a clinical setting, so called 'white coat hypertension'. If we just rely on clinic readings then such patients may be diagnosed as having hypertension when, the vast majority of the time, their blood pressure is normal.

This has led to the use of both ambulatory blood pressure monitoring (ABPM) and home blood pressure monitoring (HBPM) to confirm the diagnosis of hypertension. These techniques allow a more accurate assessment of a patients' overall blood pressure. Not only does this help prevent overdiagnosis of hypertension - ABPM has been shown to be a more accurate predictor of cardiovascular events than clinic readings.

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocate.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg

Diagnosing hypertension

Firstly, NICE recommend measuring blood pressure in both arms when considering a diagnosis of hypertension.

If the difference in readings between arms is more than 20 mmHg then the measurements should be repeated. If the difference remains > 20 mmHg then subsequent blood pressures should be recorded from the arm with the higher reading.

It should of course be remember that there are pathological causes of unequal blood pressure readings from the arms, such as supravalvular aortic stenosis. It is therefore prudent to listen to the heart sounds if a difference exists and further investigation if a very large difference is noted.

NICE also recommend taking a second reading during the consultation, if the first reading is > 140/90 mmHg. The lower reading of the two should determine further management.

NICE suggest offering ABPM or HBPM to any patient with a blood pressure >= 140/90 mmHg.

If the blood pressure is >= 180/120 mmHg:
- admit for specialist assessment if:
    - signs of retinal haemorrhage or papilloedema (accelerated hypertension) or
    - life-threatening symptoms such as new-onset confusion, chest pain, signs of heart failure, or acute kidney injury
- NICE also recommend referral if a phaeochromocytoma is suspected (labile or postural hypotension, headache, palpitations, pallor and diaphoresis)
- if none of the above then arrange urgent investigations for end-organ damage (e.g. bloods, urine ACR, ECG)
    - if target organ damage is identified, consider starting antihypertensive drug treatment immediately, without waiting for the results of ABPM or HBPM.
    - if no target organ damage is identified, repeat clinic blood pressure measurement within 7 days

Ambulatory blood pressure monitoring (ABPM)
- at least 2 measurements per hour during the person's usual waking hours (for example, between 08:00 and 22:00)
- use the average value of at least 14 measurements

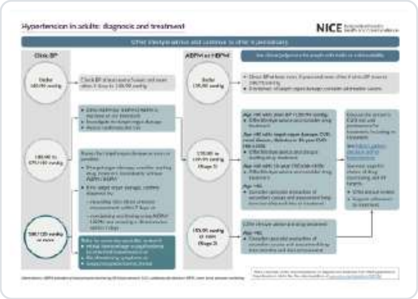
If ABPM is not tolerated or declined HBPM should be offered.

Home blood pressure monitoring (HBPM)
- for each BP recording, two consecutive measurements need to be taken, at least 1 minute apart and with the person seated
- BP should be recorded twice daily, ideally in the morning and evening
- BP should be recorded for at least 4 days, ideally for 7 days
- discard the measurements taken on the first day and use the average value of all the remaining measurements

Interpreting the results

ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)
- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)
- offer drug treatment regardless of age



Next question

Textbooks

High-yield textbook

Extended textbook

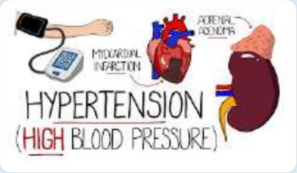
Links

NICE 👍 26 👎 37

2019 Hypertension guidelines

Suggest link Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **20.4%**

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A 24-year-old primigravida presents to the emergency department with a 72-hour history of nausea and vomiting with associated right upper quadrant pain. She as no past medical history of note and takes only pregnancy vitamins. During her 32-week midwife appointment 1 week ago, it was noted that she had gained weight and had borderline hypertension. The decision at that appointment was to monitor.

On examination, she is clinically dehydrated and appears unwell. Respiratory and cardiovascular examinations are unremarkable and abdominal examination demonstrates a gravid uterus and right upper quadrant tenderness. There is pitting oedema to the mid-shin. Observations are all within normal limits aside from a blood pressure of 145/90mmHg. She is alert and orientated, reports no headaches and has no rashes.

Blood tests are performed, results are as demonstrated:

Hb	100 g/L	Male: (135-180) Female: (115 - 160)
Platelets	97 * 10^9/L	(150 - 400)
WBC	7.3 * 10^9/L	(4.0 - 11.0)
PT	13.0 seconds	(9.5-13.5)
APTT	39.0 seconds	(30-40)
Na^+	132 mmol/L	(135 - 145)
K^+	3.4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	7.5 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
Bilirubin	45 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	350 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

An ultrasound abdomen shows patchy areas of hepatic enhanced echogenicity. Fetal monitoring is satisfactory, but the baby is in the breech position.

Given the likely diagnosis, what is the most appropriate management?

- ○ Commence IV antibiotics
- ○ Give steroids and organise an induction of labour
- ○ Organise a plasma exchange with fresh frozen plasma
- ○ Rehydrate with IV fluids and monitor
- ○ Give steroids and organise a caesarean section within 48 hours

Submit answer

Reference ranges

Score: **20.4%**

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Albumin	34 g/L	(35 - 50)

An ultrasound abdomen shows patchy areas of hepatic enhanced echogenicity. Fetal monitoring is satisfactory, but the baby is in the breech position.

Given the likely diagnosis, what is the most appropriate management?

Commence IV antibiotics	1%
Give steroids and organise an induction of labour	17%
Organise a plasma exchange with fresh frozen plasma	4%
Rehydrate with IV fluids and monitor	11%
Give steroids and organise a caesarean section within 48 hours	67%

Delivery of the baby is the treatment of HELLP syndrome

Important for me Less important

This patient has HELLP syndrome as shown by her anaemia (secondary to haemolysis), thrombocytopenia and deranged LFTs. She is 33 weeks pregnant and thus steroids would be recommended prior to delivery if possible to aid the maturation of her child's lungs. She does not have any evidence of a liver haematoma, disseminated intravascular coagulation or haemodynamic instability and thus she does not need an immediate delivery at this stage. Induction of labour would not be appropriate as her child is breech.

Differentials would include cholecystitis and thrombotic thrombocytopenic purpura (TTP).

IV antibiotics would be appropriate in the treatment of cholecystitis, but this patient has no features of infection, going against a diagnosis of cholecystitis.

Plasma exchange with fresh frozen plasma would be the recommended treatment for TTP, however, TTP would typically present with neurological symptoms such as confusion, headaches, and seizures in severe cases, which our patient does not have.

👍 👎 Discuss (8) Improve

Next question >

HELLP syndrome ★

HELLP is an acronym for Hemolysis, Elevated Liver enzymes, and a Low Platelet count. It is a serious condition that can develop in the late stages of pregnancy. Whilst there is significant overlap with severe pre-eclampsia in terms of the features some patients present with no prior history so many specialists consider it a separate entity in its own right. However, it should be remembered that around 10-20% of patients with severe preeclampsia will go on to develop HELLP.

Features
- nausea & vomiting
- right upper quadrant pain
- lethargy

Investigations
- bloods: Hemolysis, Elevated Liver enzymes, and a Low Platelet

Treatment
- delivery of the baby

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.2%**

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Question 170 of 328

A 24-year-old female presents to clinic complaining of several episodes of heart palpitations. She states that during the attacks it feels like her heart skips a bit. She denies pre-syncope or syncope. She states that she has one episode every few days. She drinks approximately 28 units of alcohol per week and several cups of coffee per day.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	100ms
QRS duration	110ms
Events	24 single ectopics with normal P wave morphology and a QRS of 110ms

What is the most appropriate initial management plan?

- ○ Bisoprolol
- ○ Ablation
- ○ Reassurance and lifestyle modifications
- ○ Implantable Cardioverter Defibrillator (ICD)
- ○ Verapamil

Submit answer

Reference ranges

Score: **20.2%**

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A 24-year-old female presents to clinic complaining of several episodes of heart palpitations. She states that during the attacks it feels like her heart skips a bit. She denies pre-syncope or syncope. She states that she has one episode every few days. She drinks approximately 28 units of alcohol per week and several cups of coffee per day.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	100ms
QRS duration	110ms
Events	24 single ectopics with normal P wave morphology and a QRS of 110ms

What is the most appropriate initial management plan?

- Bisoprolol 8%
- Ablation 8%
- Reassurance and lifestyle modifications 79%
- Implantable Cardioverter Defibrillator (ICD) 3%
- Verapamil 1%

Patients with ventricular ectopics can usually be managed by reassurance and lifestyle modifications. If pharmacological management is required, then beta blockers are first line

Important for me Less important

The ectopics captured have normal P wave and QRS duration suggestive of supraventricular ectopics of sinus or atrial origin. The first line management of supraventricular ectopics is generally reassurance and lifestyle modifications. The patient should be advised to reduce alcohol and caffeine intake. If symptoms persisted then a beta blocker would be first line.

👍 👎 💬 Discuss (8) Improve

Next question >

Beta-blockers ★

Beta-blockers are an important class of drug used mainly in the management of cardiovascular disorders.

Indications
- angina
- post-myocardial infarction
- heart failure: beta-blockers were previously avoided in heart failure but there is now strong evidence that certain beta-blockers improve both symptoms and mortality
- arrhythmias: beta-blockers have now replaced digoxin as the rate-control drug of choice in atrial fibrillation
- hypertension: the role of beta-blockers has diminished in recent years due to a lack of evidence in terms of reducing stroke and myocardial infarction.
- thyrotoxicosis
- migraine prophylaxis
- anxiety

Examples
- atenolol
- propranolol: one of the first beta-blockers to be developed. Lipid soluble therefore crosses the blood-brain barrier

Side-effects
- bronchospasm
- cold peripheries
- fatigue
- sleep disturbances, including nightmares
- erectile dysfunction

Contraindications

- uncontrolled heart failure
- asthma
- sick sinus syndrome
- concurrent verapamil use: may precipitate severe bradycardia

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 7

Beta-blockers

Suggest link Report broken link

Media

Adrenergic Antagonists

Alpha and beta blockers

Speed Pharmacology - YouTube 👍 2 👎 1

Report broken media

Score: **20.1%**

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Question 171 of 328

A 74-year-old man attends the emergency department with epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 85 beats per minute, blood pressure 135/75 mmHg, respiratory rate 16 breaths per minute, SpO2 95% (on air), and temperature 37.5ºC.

Blood results are as follows:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	88 * 10^9/L	(150 - 400)
WBC	6.2 * 10^9/L	(4.0 - 11.0)

Prothrombin time (PT)	96 secs	(10-14 secs)
INR	6.8	(<1.1)
Activated partial thromboplastin time (APTT)	44 secs	(25-35 secs)
Fibrinogen	3.2 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

- ○ Fresh frozen plasma
- ○ Intravenous 10mg vitamin K
- ○ Intravenous 3mg vitamin K
- ○ Platelet transfusion
- ○ Prothrombin complex concentrate

Submit answer

A 74-year-old man attends the emergency department with epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 85 beats per minute, blood pressure 135/75 mmHg, respiratory rate 16 breaths per minute, SpO2 95% (on air), and temperature 37.5°C.

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Prothrombin time (PT)	96 secs	(10-14 secs)
INR	6.8	(<1.1)
Activated partial thromboplastin time (APTT)	44 secs	(25-35 secs)
Fibrinogen	3.2 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

Fresh frozen plasma	3%
Intravenous 10mg vitamin K	22%
Intravenous 3mg vitamin K	69%
Platelet transfusion	0%
Prothrombin complex concentrate	7%

INR 5.0-8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, restart when INR < 5.0

Important for me Less important

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR between 5.0 - 8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In major haemorrhages, FFP should only be given if prothrombin complex concentrate is not available.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation within minutes of administration however administration of vitamin K is also required to counteract the long half-life of warfarin.

👍 👎 💬 Discuss (3) Improve

Next question >

Warfarin: management of high INR ★

The following is based on the BNF guidelines, which in turn take into account the British Committee for Standards in Haematology (BCSH) guidelines.

Situation	Management
Major bleeding (e.g. variceal haemorrhage, intracranial haemorrhage)	Stop warfarin Give intravenous vitamin K 5mg Prothrombin complex concentrate - if not available then FFP*
INR > 8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Repeat dose of vitamin K if INR still too high after 24 hours Restart warfarin when INR < 5.0
INR > 8.0 **No bleeding**	Stop warfarin Give vitamin K 1-5mg by mouth, using the intravenous preparation orally Repeat dose of vitamin K if INR still too high after 24 hours Restart when INR < 5.0
INR 5.0-8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Restart when INR < 5.0
INR 5.0-8.0 **No bleeding**	Withhold 1 or 2 doses of warfarin Reduce subsequent maintenance dose

*as FFP can take time to defrost prothrombin complex concentrate should be considered in cases of intracranial haemorrhage

+ Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 16 👎 11

Warfarin

BNF 👍 18 👎 10

Oral anticoagulants

Suggest link Report broken link

Score: **20.6%**

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Question 172 of 328

You are asked to chase up the INR result for a patient. The patient is currently admitted and being treated for a lower respiratory tract infection with clarithromycin. They take warfarin due to atrial fibrillation. The INR result is returned as below:

INR	6.8	(<1.5)

You review the patient who looks well, though they report persistent oozing of blood from where their cannula had been removed earlier that day. The gauze dressing has required changing 4 times.

What is the most appropriate course of action?

- ○ Hold warfarin and administer IV vitamin K ×
- ○ Hold warfarin and administer IV vitamin K and human prothrombin complex ×
- ○ Hold warfarin and administer oral vitamin K ×
- ○ Hold warfarin and repeat INR the next day ×
- ○ Re-prescribe warfarin at a lower dose ×

Submit answer

Reference ranges

Score: **20.6%**

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Question 172 of 328

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INR	6.8	(<1.5)

You review the patient who looks well, though they report persistent oozing of blood from where their cannula had been removed earlier that day. The gauze dressing has required changing 4 times.

What is the most appropriate course of action?

Hold warfarin and administer IV vitamin K	42%
Hold warfarin and administer IV vitamin K and human prothrombin complex	5%
Hold warfarin and administer oral vitamin K	42%
Hold warfarin and repeat INR the next day	10%
Re-prescribe warfarin at a lower dose	1%

INR 5.0-8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, restart when INR < 5.0

Important for me Less important

In this case, we have a patient with a raised INR which is likely secondary to clarithromycin use. The patient also has some minor bleeding as indicated by persistent ooze from a wound site.

Hold warfarin and administer IV vitamin K is correct. 1-3mg of IV vitamin K should be administered to this patient with minor bleeding and an INR of 5.0-8.0 as per NICE guidelines.

Hold warfarin and administer IV vitamin K and human prothrombin complex is incorrect. Human prothrombin complex should be administered in the case of major bleeding, fresh frozen plasma can be used as an alternative if human prothrombin complex is not available. As this patient is only having minimal bleeding, the use of human prothrombin complex is not recommended.

Hold warfarin and administer oral vitamin K is incorrect. This would be appropriate if the patient had no evidence of bleeding at all with an INR of >8.0. In this case, the INR is between 5.0 and 8.0 and associated with minor bleeding, intravenous vitamin K would therefore be more appropriate.

Hold warfarin and repeat INR the next day is incorrect. This would be appropriate if the INR was between 5.0 and 8.0 without any bleeding, you would then also want to reduce the maintenance dose. In this case, because there is evidence of bleeding, a dose of IV vitamin K is recommended.

Re-prescribe warfarin at a lower dose is not correct. This patient has a raised INR in the context of minor bleeding, the warfarin needs to be held and reversed in view of this.

👍 👎 Discuss (3) Improve

Next question >

Warfarin: management of high INR ★

The following is based on the BNF guidelines, which in turn take into account the British Committee for Standards in Haematology (BCSH) guidelines.

Situation	Management
Major bleeding (e.g. variceal haemorrhage, intracranial haemorrhage)	Stop warfarin Give intravenous vitamin K 5mg Prothrombin complex concentrate - if not available then FFP*

Situation	Management
INR > 8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Repeat dose of vitamin K if INR still too high after 24 hours Restart warfarin when INR < 5.0
INR > 8.0 **No bleeding**	Stop warfarin Give vitamin K 1-5mg by mouth, using the intravenous preparation orally Repeat dose of vitamin K if INR still too high after 24 hours Restart when INR < 5.0
INR 5.0-8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Restart when INR < 5.0
INR 5.0-8.0 **No bleeding**	Withhold 1 or 2 doses of warfarin Reduce subsequent maintenance dose

*as FFP can take time to defrost prothrombin complex concentrate should be considered in cases of intracranial haemorrhage

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 16 👎 11

Warfarin

BNF 👍 18 👎 10

Oral anticoagulants

Suggest link Report broken link

Score: **20.5%**

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A 26-year-old patient comes into the Emergency Department in cardiac arrest. The paramedics tell you that she was pulled out of a lake by friends after she had suddenly become unresponsive while swimming. Her friends started cardiopulmonary resuscitation (CPR) at the scene. On arrival, the paramedics noted that the patient was in Ventricular Fibrillation (VF). The patient was defibrillated, given IV amiodarone and adrenaline. Her friends tell you she has no significant past medical history and was always fit and well until this point.

Despite continued attempts to resuscitate the patient, she dies in the Emergency Department. A post-mortem is carried out which is normal.

What is the most likely diagnosis?

○ Hypertrophic Obstructive Cardiomyopathy (HOCM) ×

○ Long QT1 Syndrome ×

○ Long QT3 Syndrome ×

○ Right Arrhythmogenic Ventricular Dysplasia (RAVD) ×

○ Wolf-Parkinson White Syndrome ×

Submit answer

Reference ranges

Score: **20.5%**

1 ×

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Question 173 of 328 ✓

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Despite continued attempts to resuscitate the patient, she dies in the Emergency Department. A post-mortem is carried out which is normal.

What is the most likely diagnosis?

Hypertrophic Obstructive Cardiomyopathy (HOCM)	15%
Long QT1 Syndrome	53%
Long QT3 Syndrome	16%
Right Arrhythmogenic Ventricular Dysplasia (RAVD)	8%
Wolf-Parkinson White Syndrome	7%

All of these differentials can result in sudden cardiac death in a young patient. However, you have been told here that the patient's postmortem was normal which makes HOCM and RAVD unlikely.

This leaves Long QT1 Syndrome, Long QT3 Syndrome and Wolf-Parkinson-White as possible differentials. The big clue here is that the patient became unresponsive when swimming which is typical of Long QT1 Syndrome.

Long QT syndromes can result in sudden cardiac death when patients are exposed to an adrenergic surge which can put the patient into VF or VT.

Long QT1: adrenergic surge due to intense physical activity such as swimming

Long QT2: adrenergic surge due to intense emotion such as excitement or fear

Long QT3: death during sleep

👍 👎 Discuss (4) Improve

Next question >

Long QT syndrome

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) • methadone • chloroquine • terfenadine** • erythromycin • haloperidol	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Congenital	Drugs*	Other
	• ondanestron	

Features

- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management

- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

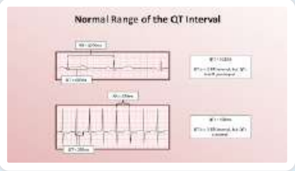
2010 Drug-Induced Long QT syndrome

Life in the Fast Lane 👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link Report broken link

Media



Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **20.9%**

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Question 174 of 328

You are asked to review a 22-year-old female with a heart murmur. Her height is 4ft 7 inches and she reports having always been short for her year. She has been diagnosed with autoimmune hypothyroidism, for which she is taking levothyroxine replacement and is also taking 'hormone replacement to protect her bones' after her GP noted she had not started her periods by 19 years old.

On examination, you note short 4th metacarpals on both hands and lymphoedematous in her hands and feet. Auscultation of her precordium reveals an ejection systolic murmur at the left infraclavicular region. Her body mass index is 22.4 kg/m². The patient's blood tests are as follows:

Hb	123 g/l
Platelets	380 * 10^9/l
WBC	6.5 * 10^9/l

Na^+	144 mmol/l
K^+	4.8 mmol/l
Urea	7.2 mmol/l
Creatinine	110 µmol/l
Adjusted calcium	2.40 mmol/l
Phosphate	1.1 mmol/l
PTH	9 mg/dl (8.5-10.2mg/dl)

What is the underlying unifying diagnosis?

- ○ Turner's syndrome ×
- ○ Pseudohypoparathyroidism ×
- ○ Pseudopseudohypoparathyroidism ×
- ○ Noonan syndrome ×

○ Down's syndrome ×

Submit answer

Reference ranges ⌄

Score: **20.9%**

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Question 174 of 328 ✓

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Phosphate	1.1 mmol/l
PTH	9 mg/dl (8.5-10.2mg/dl)

What is the underlying unifying diagnosis?

Turner's syndrome	59%
Pseudohypoparathyroidism	17%
Pseudopseudohypoparathyroidism	12%
Noonan syndrome	9%

Down's syndrome 2%

A syndromic presentation appears most likely in this patient. An acute infective endocarditis is unlikely for someone who is systemically well, a regurgitant murmur would also be more likely. The symptoms cannot be attributed to delayed puberty, defined as onset of puberty outside 95% of the normal population and typically before 18 years old. The combination of primary amenorrhoea, short stature, lymphoedema, shortened 4th metacarpal, autoimmune predisposition and an ejection systolic murmur suggestive of aortic coarctation should infer a diagnosis of Turner's syndrome, a disorder caused by loss of an X chromosome.

Noonan syndrome, an autosomal dominant disorder, is a reasonable differential in a patient with short stature, lymphoedema and cardiac problems. However, patients with Noonan syndrome typically presents with learning difficulties, facial dysmorphisms and webbed neck. While valvular abnormalities are less common, hypertrophic cardiomyopathy is more commonly present.

Similar to Noonan syndrome, Down's syndrome, caused by trisomy 21, also classically presents with facial dysmorphisms such as upslanting palpebral fissures, low-set ears and epicanthic folds. Cognitive impairment and learning difficulties are also common. However, similar to our patient, short stature, thyroid dysfunction and cardiac problems, predominantly ASD, VSD and PDA are frequently associated.

Pseudohypoparathyroidism is caused by normal parathyroid hormone release but end organ resistance to hormonal effects, resulting classically in round facies and shortened fourth and fifth metacarpals. The patient is commonly obese with possible learning difficulties.

Pseudopseudohypoparathyroidism presents with the phenotype of pseudohypoparathyroidism but with normal calcium chemistry.

👍 👎 Discuss (6) Improve

Next question >

Turner's syndrome ★

Turner's syndrome is a chromosomal disorder affecting around 1 in 2,500 females. It is caused by either the presence of only one sex chromosome (X) or a deletion of the short arm of one of the X chromosomes. Turner's syndrome is denoted as 45,XO or 45,X.

Features
- short stature
- shield chest, widely spaced nipples
- webbed neck
- bicuspid aortic valve (15%), coarctation of the aorta (5-10%)
    - an increased risk of aortic dilatation and dissection are the most serious long-term health problems for women with Turner's syndrome
    - regular monitoring in adult life for these complications is an important component of care
- primary amenorrhoea
- cystic hygroma (often diagnosed prenatally)
- high-arched palate
- short fourth metacarpal
- multiple pigmented naevi
- lymphoedema in neonates (especially feet)
- gonadotrophin levels will be elevated
- hypothyroidism is much more common in Turner's
- horseshoe kidney: the most common renal abnormality in Turner's syndrome

There is also an increased incidence of autoimmune disease (especially autoimmune thyroiditis) and Crohn's disease

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

TURNER SYNDROME
Osmosis.org

Turner's syndrome

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **21.4%**

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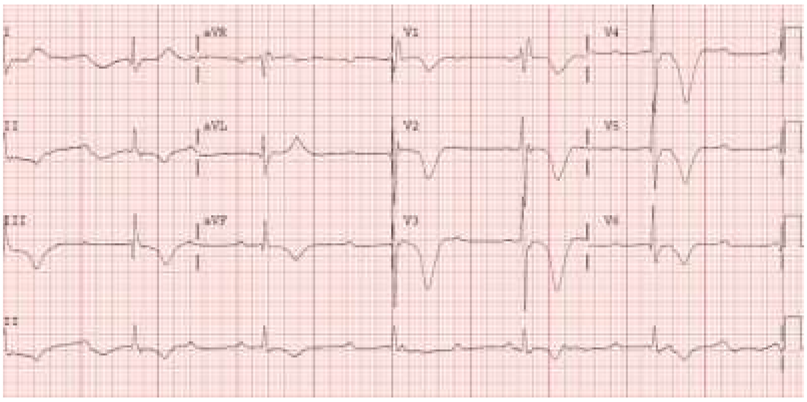
Question 175 of 328

A 56-year-old man is brought to the Emergency Department. He works as a window cleaner. His colleague describes him suddenly falling from the ladder without warning. On admission he is conscious with a bradycardia of around 36/min. His blood pressure is 110/62 mmHg and he is well perfused with no signs of heart failure. An ECG is taken on account of his bradycardia:



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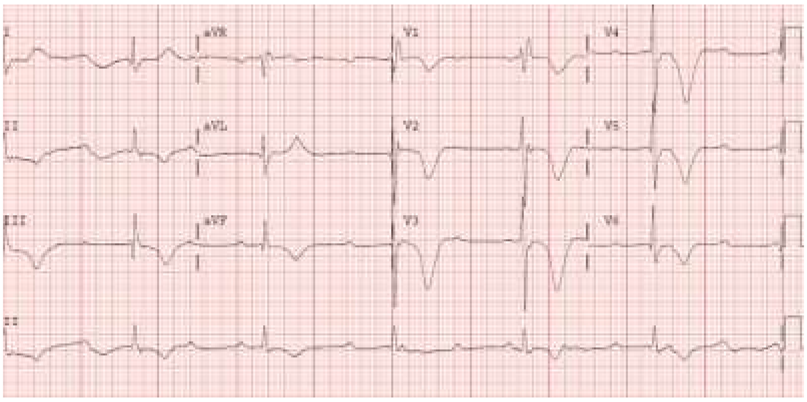
A previous ECG taken four months ago was completely normal. What is the most likely diagnosis?

- ○ Sick sinus syndrome
- ○ Stokes-Adams attack with associated complete heart block
- ○ Myocardial infarction with new left bundle branch block
- ○ Second-degree atrioventricular block (Mobitz II)
- ○ Second-degree atrioventricular block (Mobitz I / Wenckebach)

Submit answer

Reference ranges

A 56-year-old man is brought to the Emergency Department. He works as a window cleaner. His colleague describes him suddenly falling from the ladder without warning. On admission he is conscious with a bradycardia of around 36/min. His blood pressure is 110/62 mmHg and he is well perfused with no signs of heart failure. An ECG is taken on account of his bradycardia:



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A previous ECG taken four months ago was completely normal. What is the most likely diagnosis?

Sick sinus syndrome	4%
Stokes-Adams attack with associated complete heart block	53%
Myocardial infarction with new left bundle branch block	5%
Second-degree atrioventricular block (Mobitz II)	16%
Second-degree atrioventricular block (Mobitz I / Wenckebach)	22%

There are regular p waves at a rate of around 90 / min but they do not conduct. The ventricular escape rhythm has a rate of about 36 / min and has a right bundle branch block (RBBB) like morphology. The bizarre, wide, inverted T-waves can be seen in Stokes-Adams attacks and do not necessarily imply new ischaemia.

👍 | 👎 | Discuss (5) | Improve

Next question >

Complete heart block ★

Features

- syncope
- heart failure
- regular bradycardia (30-50 bpm)
- wide pulse pressure
- JVP: cannon waves in neck
- variable intensity of S1

Types of heart block

First degree heart block

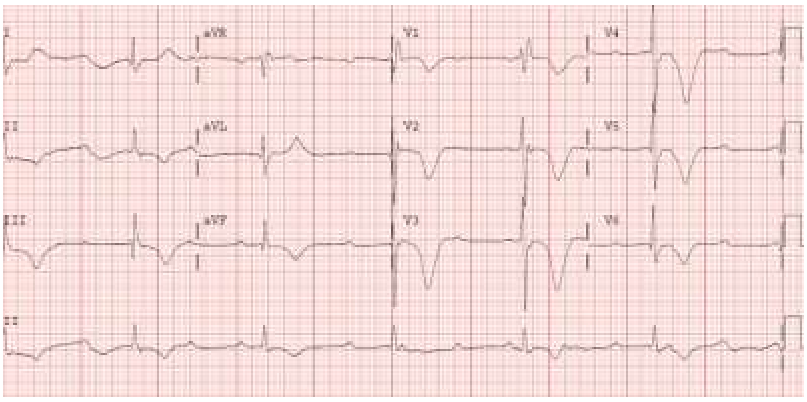
- PR interval > 0.2 seconds

Second degree heart block

- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex

Third degree (complete) heart block

- there is no association between the P waves and QRS complexes



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ECG showing third degree (complete) heart block

123

Next question >

Textbooks

High-yield textbook

Extended textbook

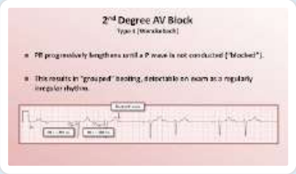
Links

ECGPedia 👍 13 👎 10

AV conduction problems

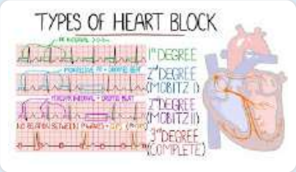
Suggest link Report broken link

Media


2nd Degree AV Block

AV block

Strong Medicine - YouTube 👍 2 👎 0


TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Atrioventricular block

Osmosis - YouTube 👍 0 👎 1

Report broken media

Score: **21.3%**

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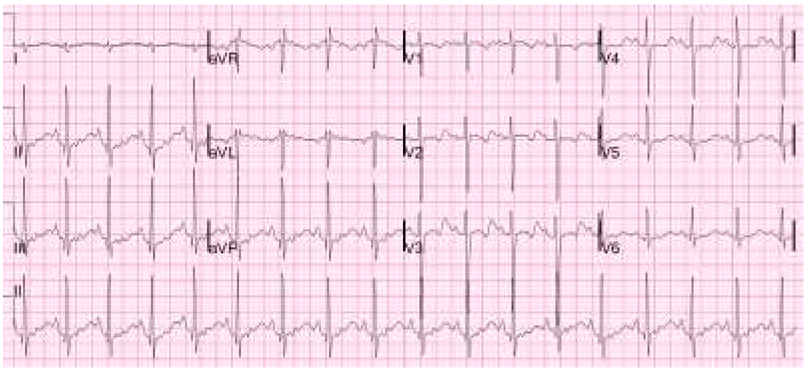
Question 176 of 328

A 22-year-old woman presents to the emergency department with syncope. She also states that he has had profuse vomiting and diarrhoea for the last 3 days. Observations are as follows: heart rate 100 bpm, blood pressure 100/60 mmHg, respiratory rate 18 breaths per minute, SpO2 98% on air, temperature 37.2 ºC.

An ECG is performed in the department:



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What is the likely cause of this ECG abnormality?

- ○ Arrhythmogenic right ventricular dysplasia (ARVD) ×
- ○ Hyperkalaemia ×
- ○ Hypokalaemia ×
- ○ Hypothermia ×
- ○ Wolff-Parkinson-White syndrome ×

Submit answer

Reference ranges

Score: **21.3%**

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A 22-year-old woman presents to the emergency department with syncope. She also states that he has had profuse vomiting and diarrhoea for the last 3 days. Observations are as follows: heart rate 100 bpm, blood pressure 100/60 mmHg, respiratory rate 18 breaths per minute, SpO2 98% on air, temperature 37.2 ºC.

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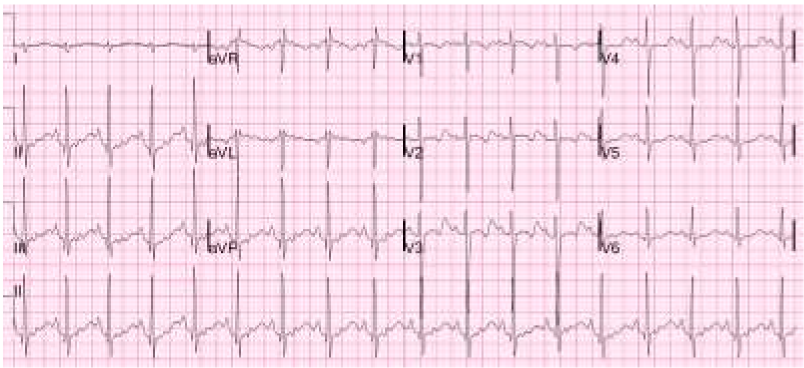

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What is the likely cause of this ECG abnormality?

Arrhythmogenic right ventricular dysplasia (ARVD)	18%
Hyperkalaemia	4%
Hypokalaemia	71%
Hypothermia	3%
Wolff-Parkinson-White syndrome	5%

The ECG demonstrates sinus rhythm with a rate of approximately 100 beats per minute. There are prominent U waves following the T wave.

Hypokalaemia is correct. The electrocardiographic features are most in keeping with hypokalaemia. U waves are present and classic of hypokalemia, however, it is important to remember that they can be present in other conditions (e.g. bradycardia, hypocalcaemia, hypomagnesaemia, hypothermia and raised intracranial pressure). Other ECG features of hypokalaemia include small or absent T waves (occasionally inversion), prolonged PR interval, ST depression, and long QTc.

Arrhythmogenic right ventricular dysplasia (ARVD) is incorrect. ARVD is characterised by an epsilon wave which is a small deflection buried at the end of the QRS complex usually best seen in the ST segment of V1 and V2.

Hyperkalaemia is incorrect. ECG features of hyperkalaemia include peaked T waves, P wave widening/flattening, PR prolongation, bradyarrhythmias, and QRS widening with bizarre QRS morphology.

Hypothermia is incorrect. Hypothermia classically presents with sinus bradycardia and Osborn waves (J wave) defined as positive deflection seen at the J point in precordial and true limb leads. Osborn waves are characteristically seen in hypothermia, however, they are not pathognomonic and can be seen in other conditions such as hypercalcaemia and myocarditis.

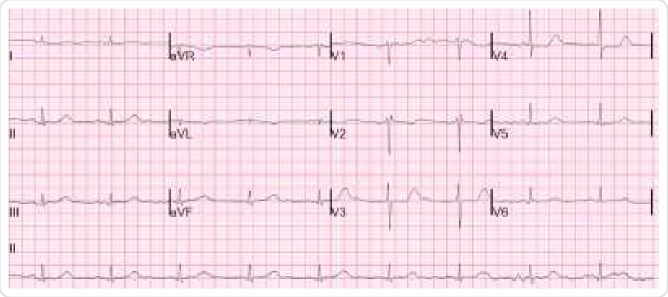
Wolff-Parkinson-White syndrome is incorrect. This condition is associated with a short PR interval (< 120ms), broad QRS (> 100ms), and a slurred upstroke to the QRS complex (the delta wave).

👍 👎 Discuss (2) Improve

Next question ›

ECG: hypokalaemia ★

ECG features of hypokalaemia
- U waves
- small or absent T waves (occasionally inversion)
- prolong PR interval
- ST depression
- long QT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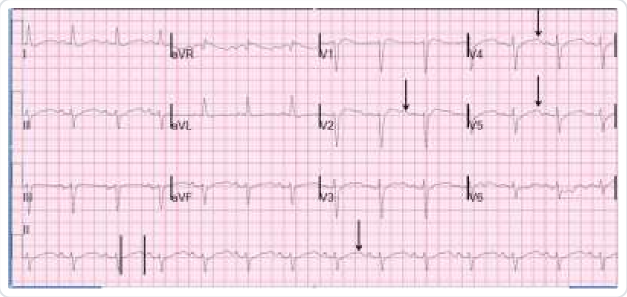
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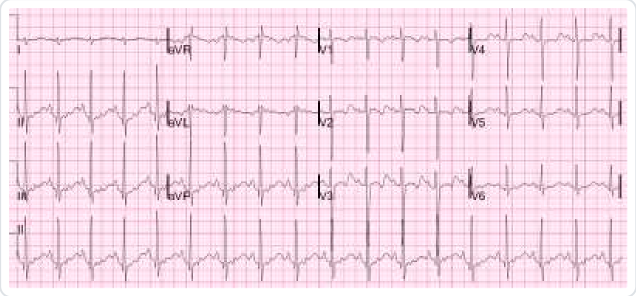
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In Hypokalaemia, U have no Pot and no T, but a long PR and a long QT

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 48 👎 6

ECG: Hypokalaemia changes

Suggest link Report broken link

Score: **21.1%**

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A 19-year-old woman presents to the acute medical take with three days of acute shortness of breath. She initially had a dry cough and sore throat but became increasingly lethargic. She is usually fit and well and is fully vaccinated. She is taking the combined oral contraceptive pill. On examination, she had bibasal crepitations with dullness to percussion at the bases. Her observations show a respiratory rate of 25 breaths per minute, heart rate 105 beats per minute and saturations of 96% maintained on 60% venturi mask. Her JVP is seen at 5 cm above her sternal angle and there are no murmurs.

Na^+	130 mmol/l
K^+	4.9 mmol/l
Urea	5.6 mmol/l
Creatinine	90 µmol/l
Troponin	20,000 ng/ml (normal range <20)
ECG	ST elevation seen in V1-V4

What is the most likely diagnosis?

○ Myocarditis ×

○ Myxoedema coma ×

○ Nephrotic syndrome ×

○ Myocardial infarction ×

○ Takotsubo cardiomyopathy ×

Submit answer

Reference ranges

Question 177 of 328

A 19-year-old woman presents to the acute medical take with three days of acute shortness of breath. She initially had a dry cough and sore throat but became increasingly lethargic. She is usually fit and well and is fully vaccinated. She is taking the combined oral contraceptive pill. On examination, she had bibasal crepitations with dullness to percussion at the bases. Her observations show a respiratory rate of 25 breaths per minute, heart rate 105 beats per minute and saturations of 96% maintained on 60% venturi mask. Her JVP is seen at 5 cm above her sternal angle and there are no murmurs.

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K^+	4.9 mmol/l
Urea	5.6 mmol/l
Creatinine	90 µmol/l
Troponin	20,000 ng/ml (normal range <20)
ECG	ST elevation seen in V1-V4

What is the most likely diagnosis?

Myocarditis	77%
Myxoedema coma	1%
Nephrotic syndrome	1%
Myocardial infarction	8%
Takotsubo cardiomyopathy	13%

Myocarditis presents with ST elevation and acute pulmonary oedema in a young patient with a recent flu-like illness

Important for me Less important

Bibasal crepitations and a raised JVP show acute heart failure. She has no chest pain making an acute myocardial infarction unlikely and there is no recent emotional distress as would be in Takotsubo cardiomyopathy. Given her recent viral illness, this is the most likely cause of her sudden deterioration in someone who was formerly well. Nephrotic syndrome and myxoedema coma would not have ST elevation.

👍 👎 Discuss (5) Improve

Next question ›

Myocarditis ★

Myocarditis describes inflammation of the myocardium. There are a wide range of underlying causes. It should be particularly considered in younger patients who present with chest pain.

Causes
- viral: coxsackie B, HIV
- bacteria: diphtheria, clostridia
- spirochaetes: Lyme disease
- protozoa: Chagas' disease, toxoplasmosis
- autoimmune
- drugs: doxorubicin

Presentation
- usually young patient with an acute history
- chest pain
- dyspnoea
- arrhythmias

Investigations
- bloods
    - ↑ inflammatory markers in 99%
    - ↑ cardiac enzymes
    - ↑ BNP
- ECG
    - tachycardia
    - arrhythmias

    - ST/T wave changes including ST-segment elevation and T wave inversion

Management
- treatment of underlying cause e.g. antibiotics if bacterial cause
- supportive treatment e.g. of heart failure or arrhythmias

Complications
- heart failure
- arrhythmia, possibly leading to sudden death
- dilated cardiomyopathy: usually a late complication

+ Q 123

Next question >

Textbooks

High-yield textbook

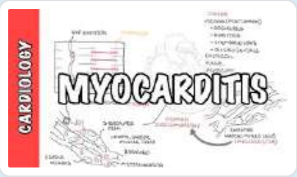
Extended textbook

Media

MYOCARDITIS
Osmosis.org

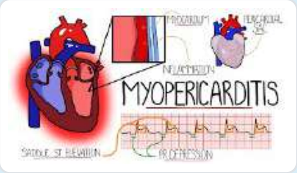
Myocarditis

Osmosis - YouTube 8 0


CARDIOLOGY
MYOCARDITIS

Myocarditis

Armando Hasudungan - YouTube 👍 3 👎 0


MYOPERICARDITIS

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **21%**

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Question 178 of 328

A 58-year-old woman was admitted with a stroke following a month's history of recurrent fevers, anorexia and weight loss. On examination, she had a left-sided hemiparesis and facial droop. Cardiovascular examination revealed splinter haemorrhages in 5 of her fingers across both hands, and a soft diastolic murmur heard loudest in expiration over the aortic area.

A trans-thoracic echocardiogram showed an oscillating vegetation on an aortic leaflet, in the path of regurgitant jets. Two blood cultures were positive for Streptococci spp. She was diagnosed with infective endocarditis and started on intravenous benzylpenicillin 1.2g every 4 hours and gentamicin 1mg/kg twice daily therapy.

She was reviewed after 5 days on antibiotic therapy with the following results.

Na+	139 mmol/l
K+	4.7 mmol/l
Urea	14.2 mmol/l
Creatinine	178 µmol/l
Serum bilirubin	16 µmol/l
Serum alkaline phosphatase	115 IU/l
Serum aspartate aminotransferase	18 IU/l
C Reactive protein	89 mg/l
Haemoglobin	138 g/l
White cell count	13.6 x 10^9/L
INR	1.1
Blood cultures	*Streptococcus Bovis*
Penicillin Minimum Inhibitory Concentration (MIC)	0.6 mg/L (high)
ECG	Prolonged PR interval (not present on admission ECG)

What is the next most important management?

- ○ Increase antibiotics to intravenous benzylpenicillin 2.4g every 4 hours and gentamicin 1mg/kg twice daily ×
- ○ Organise an urgent colonoscopy ×
- ○ Switch antibiotics to ceftriaxone 2g once daily ×
- ○ Refer to cardiothoracic surgeons ×
- ○ Organise urgent trans-oesophageal echocardiogram ×

Submit answer

Reference ranges ⌄

Score: **21%**

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178 -

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What is the next most important management?

Increase antibiotics to intravenous benzylpenicillin 2.4g every 4 hours and gentamicin 1mg/kg twice daily	3%
Organise an urgent colonoscopy	39%
Switch antibiotics to ceftriaxone 2g once daily	3%
Refer to cardiothoracic surgeons	46%
Organise urgent trans-oesophageal echocardiogram	9%

This patient has infective endocarditis, as diagnosed by two positive major criteria from the Duke's criteria for infective endocarditis. In addition, she has a number of minor criteria, including fever, vascular events (stroke), immunological events (splinter haemorrhages, renal impairment secondary to glomerulonephritis). Despite starting on appropriate empirical treatment she appears to have an ongoing infection from her blood tests, and her ECG demonstrates a prolonged PR interval which may be reflective of an enlarging aortic abscess disrupting the atrioventricular node, which is an indication for referral for cardiothoracic surgery in infective endocarditis.

Other surgical indications include

- Heart failure: valve obstruction resulting in pulmonary oedema or shock, severe acute regurgitation
- Uncontrolled infection: abscess, false aneurysm, persisting fever and positive blood cultures for greater than 10 days despite appropriate antibiotics, multiresistant microorganisms
- Prevention of embolism: large vegetations resulting in one or more embolic episodes despite appropriate antibiotic therapy, or other predictors of complications eg. heart failure.

She has a penicillin-resistant streptococci, according to the British Society of Antimicrobial Chemotherapy guidelines, should be treated with vancomycin and gentamicin, so options (a) and (c) are incorrect. *Streptococcus Bovis* bacteraemia is associated with bowel malignancy, and a colonoscopy should be arranged to rule out malignancy, however, the worsening PR prolongation takes priority and should be managed first.

An urgent trans-oesophageal echocardiogram may be useful to evaluate the size of the aortic abscess, however, it would not change management as this patient needs to be referred to the cardiothoracic surgeons as there is already evidence of enlarging vegetation.

👍 👎 Discuss (16) Improve

Next question

Infective endocarditis: prognosis and management

Poor prognostic factors
- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism
- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin If prosthetic valve

Scenario	**Suggested antibiotic therapy**
	- vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA - vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA - vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic - vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic - vancomycin + low-dose gentamicin

Indications for surgery
- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

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Score: **20.9%**

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A 75-year-old man is an inpatient on the orthopaedic ward recovering from an elective knee replacement performed three days previously. The patient's immediate post-operative recovery had been unremarkable and he had begun to mobilise with the ward physiotherapist. Routine observations recorded on the ward had been unremarkable during the previous 24 hours.

During her drug round, the patient's nurse saw the patient suddenly become unable to breathe and clutch at his chest. After calling for help the nurse went to her patient and found him to be in cardiac arrest. Resuscitations attempts were initiated following advanced life-support protocol. Please see the below table for a summary of the patient's electrical rhythm and treatments administered during the initial phases of the resuscitation attempt.

The patient was noted to be in good physical health with his only comorbidity being hypertension, well controlled with medication. The patient's admission clerking recorded that he was a retired schoolteacher who lived independently at home with his wife. A review of the patient's drug chart indicated that he had been receiving subcutaneous enoxaparin as prophylaxis against venous thromboembolism, but that the patient had refused to wear compression stockings during his admission as he found them uncomfortable.

Number of rhythm check	Result of rhythm check	Treatment administered
1	Pulseless electrical activity	IV adrenaline 1 mg
2	Ventricular fibrillation (coarse)	DC shock 150 J
3	Ventricular fibrillation	???

In addition to a further DC shock, what is the appropriate choice of IV drug treatment following the third rhythm check?

○ IV adrenaline 1 mg ×

○ IV atropine 400 micrograms & IV amiodarone 300 mg ×

○ No IV drug treatment indicated ×

○ IV adrenaline 1 mg & IV amiodarone 300 mg ×

○ IV atropine 400 micrograms ×

Submit answer

Reference ranges ∨

Score: **20.9%**

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A 75-year-old man is an inpatient on the orthopaedic ward recovering from an elective knee replacement performed three days previously. The patient's immediate post-operative recovery had been unremarkable and he had begun to mobilise with the ward physiotherapist. Routine observations recorded on the ward had been unremarkable during the previous 24 hours.

During her drug round, the patient's nurse saw the patient suddenly become unable to breathe and clutch at his chest. After calling for help the nurse went to her patient and found him to be in cardiac arrest. Resuscitations attempts were initiated following advanced life-support protocol. Please see the below table for a summary of the patient's electrical rhythm and treatments administered during the initial phases of the resuscitation attempt.

The patient was noted to be in good physical health with his only comorbidity being hypertension, well controlled with medication. The patient's admission clerking recorded that he was a retired schoolteacher who lived independently at home with his wife. A review of the patient's drug chart indicated that he had been receiving subcutaneous enoxaparin as prophylaxis against venous thromboembolism, but that the patient had refused to wear compression stockings during his admission as he found them uncomfortable.

Number of rhythm check	Result of rhythm check	Treatment administered
1	Pulseless electrical activity	IV adrenaline 1 mg
2	Ventricular fibrillation (coarse)	DC shock 150 J
3	Ventricular fibrillation	???

In addition to a further DC shock, what is the appropriate choice of IV drug treatment following the third rhythm check?

IV adrenaline 1 mg	24%
IV atropine 400 micrograms & IV amiodarone 300 mg	2%
No IV drug treatment indicated	15%
IV adrenaline 1 mg & IV amiodarone 300 mg	59%
IV atropine 400 micrograms	0%

The advanced life support algorithm for pulseless electrical activity (PEA) and asystole requires adrenaline to be given immediately and then continued every 3 to 5 minutes (in practice, after every second rhythm check). By contrast, the algorithm for ventricular fibrillation and ventricular tachycardia (VF / VT) requires IV adrenaline to not be given until after the third shock and then continued every 3 to 5 minutes.

However, once adrenaline has been given during a resuscitation attempt it should be given every 3-5 minutes even if the type of rhythm changes from PEA/asystole to VF / VT, even if this is only the second cycle of the rhythm being VF / VT (as in the example in the question).

As part of the VF / VT algorithm, IV amiodarone 300 mg should be given after three defibrillation attempts. A further dose of IV amiodarone 150 mg should be considered after a total of five defibrillation attempts. Atropine is not used as part of the advanced life-support algorithm.

https://www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/

Discuss (6) Improve

Next question ›

Adult advanced life support ★

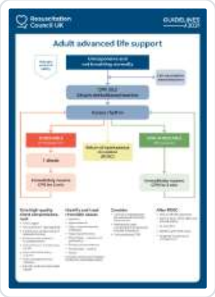
The following is based on the 2021 Resus Council guidelines. Please see the link for more details, below is only a very brief summary of key points.

It should be remembered that the algorithm divides patients into those with:

- 'shockable' rhythms: ventricular fibrillation/pulseless ventricular tachycardia (VF/pulseless VT)
- 'non-shockable' rhythms: asystole/pulseless-electrical activity (asystole/PEA)

Major points include:

- chest compressions
  - the ratio of chest compressions to ventilation is 30:2
  - chest compressions are now continued while a defibrillator is charged
- defibrillation
  - a single shock for VF/pulseless VT followed by 2 minutes of CPR
  - if the cardiac arrested is witnessed in a monitored patient (e.g. in a coronary care unit) then the 2015 guidelines recommend 'up to three quick successive (stacked) shocks', rather than 1 shock followed by CPR
- drug delivery
  - IV access should be attempted and is first-line
  - if IV access cannot be achieved then drugs should be given via the intraosseous route (IO)
  - delivery of drugs via a tracheal tube is no longer recommended
- adrenaline
  - adrenaline 1 mg as soon as possible for non-shockable rhythms
  - during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock
  - repeat adrenaline 1mg every 3-5 minutes whilst ALS continues
- amiodarone
  - amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.
  - a further dose of amiodarone 150 mg should be given to patients who are in VF/pulseless VT after 5 shocks have been administered
  - lidocaine used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead
- thrombolytic drugs
  - should be considered if a pulmonary embolus is suspected
  - if given, CPR should be continued for an extended period of 60-90 minutes



Other points

- atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA)
- following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

Reversible causes of cardiac arrest:

The 'Hs'	The 'Ts'
• Hypoxia • Hypovolaemia • Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders • Hypothermia	• Thrombosis (coronary or pulmonary) • Tension pneumothorax • Tamponade - cardiac • Toxins

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council 👍 32 👎 14

2021 Basic Life Support guidelines

Resuscitation Council 👍 58 👎 25

2021 Advanced Life Support guidelines

Suggest link Report broken link

Media

RC (UK) Cardiac Arrest Management Demo

Resuscitation Council (UK) - YouTube 👍 16 👎 2

Report broken media

Score: **20.8%**

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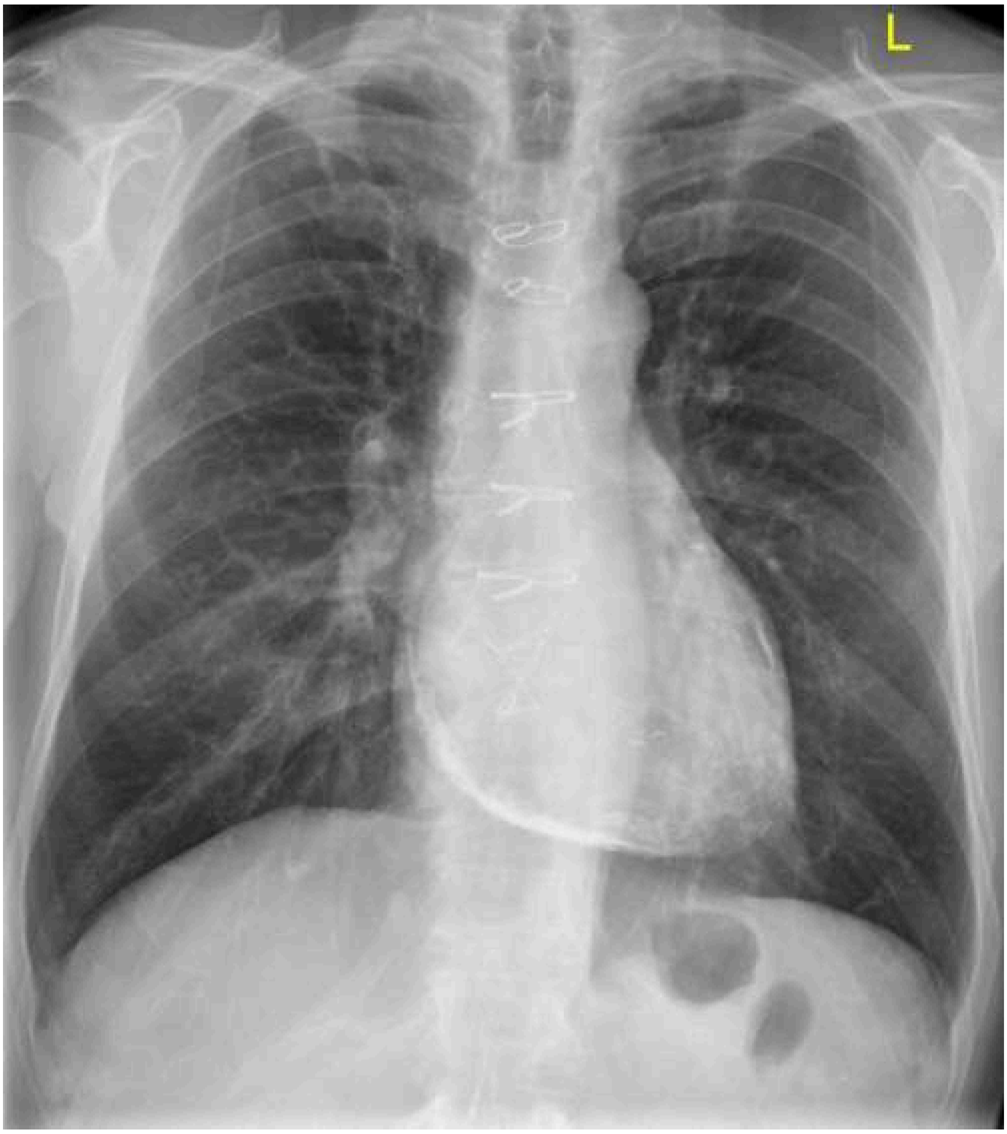
A 60-year-old man is admitted to the Emergency Department with acute dyspnoea. He is unable to give a full history and his notes are not yet available.

His chest x-ray is shown below:



© Image used on license from Radiopaedia

What is the most likely explanation for these changes seen over the heart?

○ Left ventricular aneurysm ×

○ Sarcoidosis ×

○ Atrial myxoma ×

○ Primary hyperparathyroidism ×

○ Previous episodes of uraemia ×

Submit answer

Reference ranges ⌵

Score: **20.8%**

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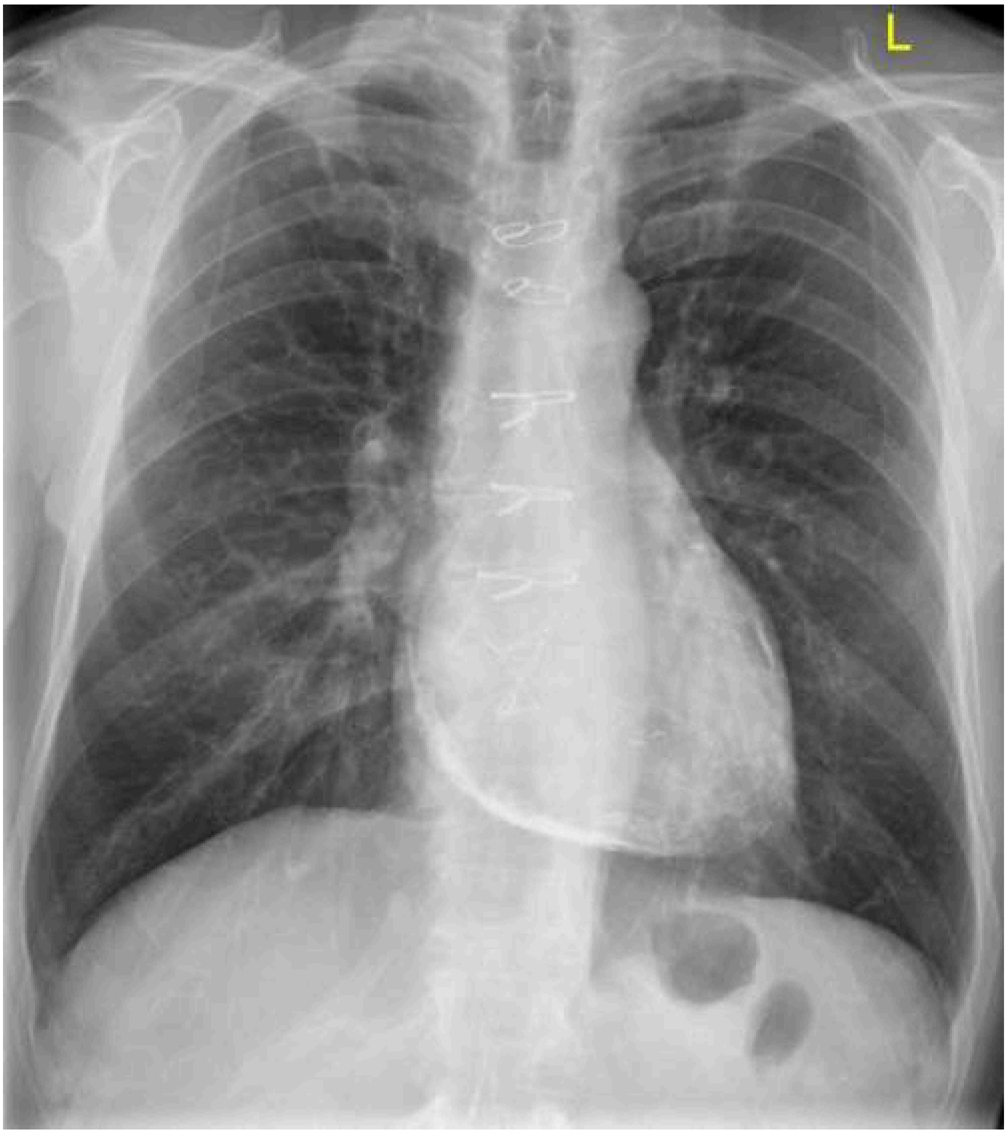
Question 180 of 328

A 60-year-old man is admitted to the Emergency Department with acute dyspnoea. He is unable to give a full history and his notes are not yet available.

His chest x-ray is shown below:



© Image used on license from Radiopaedia

What is the most likely explanation for these changes seen over the heart?

Left ventricular aneurysm 18%

Sarcoidosis 21%

Atrial myxoma 10%

Primary hyperparathyroidism 11%

Previous episodes of uraemia 41%

Pericardial calcification most commonly develops following repeated episodes of acute pericarditis. Calcification of the pericardium often results in constrictive pericarditis.

👍 👎 Discuss (11) Improve

Next question >

Acute pericarditis ★

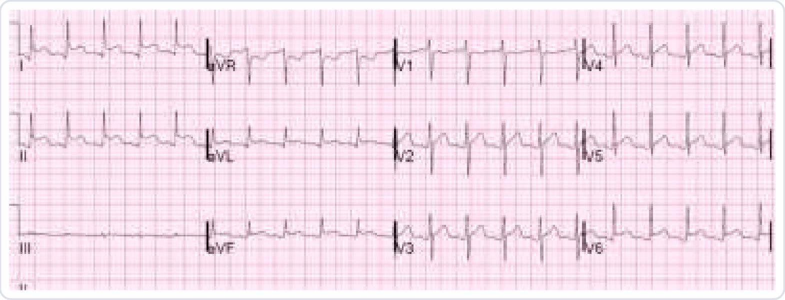
Acute pericarditis is a condition referring to inflammation of the pericardial sac, lasting for less than 4-6 weeks.

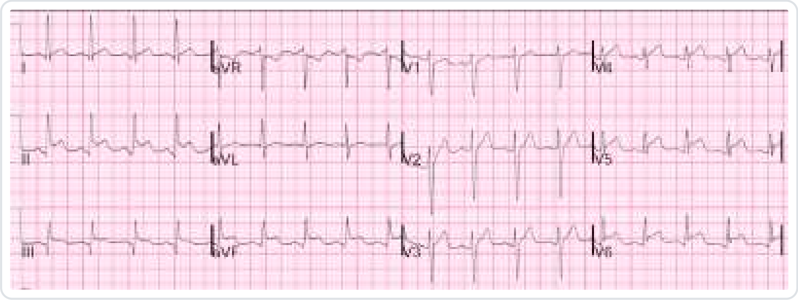
Aetiology
- viral infections (Coxsackie)
- tuberculosis
- uraemia
- post-myocardial infarction
  - early (1-3 days): fibrinous pericarditis
  - late (weeks to months): autoimmune pericarditis (Dressler's syndrome)
- radiotherapy
- connective tissue disease
  - systemic lupus erythematosus
  - rheumatoid arthritis
- hypothyroidism
- malignancy
  - lung cancer
  - breast cancer
- trauma

Features
- chest pain: may be pleuritic. Is often relieved by sitting forwards

- other symptoms include a non-productive cough, dyspnoea and flu-like symptoms
- pericardial rub

Investigations
- ECG changes
    - the changes in pericarditis are often global/widespread, as opposed to the 'territories' seen in ischaemic events
    - 'saddle-shaped' ST elevation
    - PR depression: most specific ECG marker for pericarditis
- all patients with suspected acute pericarditis should have transthoracic echocardiography
- bloods
    - inflammatory markers
    - troponin: around 30% of patients may have an elevated troponin - this indicates possible myopericarditis





Management
- the majority of patients can be managed as outpatients
    - patients who have high-risk features such as fever > 38°C or elevated troponin should be managed as an inpatient
- treat any underlying cause
    - most patients however will have pericarditis secondary to viral infection, meaning no specific treatment is indicated
- strenuous physical activity should be avoided until symptom resolution and normalisation of inflammatory markers
- a combination of NSAIDs and colchicine is now generally used for first-line for patients with acute idiopathic or viral pericarditis

    - until symptom resolution and normalisation of inflammatory markers (usually 1-2 weeks) followed by tapering of dose recommended over

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 35 👎 15

Pericarditis review

Suggest link Report broken link

Media

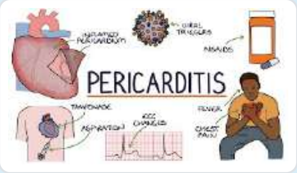
Pericardial rub - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 0

PERICARDITIS & PERICARDIAL EFFUSIONS
Osmosis.org

Pericarditis and pericardial effusions

Osmosis - YouTube 👍 2 👎 0


PERICARDITIS

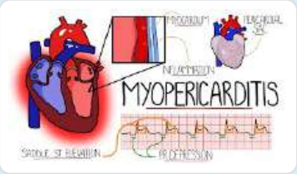
Pericarditis

Zero To Finals - YouTube 👍 0 👎 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 👍 2 👎 1


MYOPERICARDITIS

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **20.7%**

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Question 181 of 328

A 19-year-old female presents to the medical outpatient with a history of palpitations. She suffers from bouts of anxiety and dizziness associated with these palpitations and has had one episode of syncope. She has had bouts of atrial fibrillation in the past, although documentary evidence is not available.

On examination, her blood pressure is 125/85 mmHg and her pulse is 140 bpm.

Her ECG reveals a broad complex regular tachycardia with a short PR interval and a slurred upstroke of the QRS complex. There is additionally a tall R wave in V1.

Which of the following would be the most appropriate initial step in medical management?

- ○ IV adenosine ×
- ○ IV verapamil ×
- ○ IV digoxin ×
- ○ IV propranolol ×
- ○ IV procainamide ×

Submit answer

Reference ranges

Score: **20.7%**

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A 19-year-old female presents to the medical outpatient with a history of palpitations. She suffers from bouts of anxiety and dizziness associated with these palpitations and has had one episode of syncope. She has had bouts of atrial fibrillation in the past, although documentary evidence is not available.

On examination, her blood pressure is 125/85 mmHg and her pulse is 140 bpm.

Her ECG reveals a broad complex regular tachycardia with a short PR interval and a slurred upstroke of the QRS complex. There is additionally a tall R wave in V1.

Which of the following would be the most appropriate initial step in medical management?

IV adenosine	11%
IV verapamil	12%
IV digoxin	2%
IV propranolol	7%
IV procainamide	67%

The description of the ECG given is that of WPW syndrome, a form of Atrio-Ventricular Reciprocating Tachycardia that results from conduction over an accessory pathway.

There are two types of WPW syndrome, Type A and Type B.

In case of type A (left atrioventricular connections), a positive R wave will be seen in V1 ('positive delta') on the precordial leads of the electrocardiogram,

In type B (right atrioventricular connections), a predominantly negative delta wave will be seen in lead V1 ('negative delta').

The treatment of choice in the long term management is radiofrequency catheter ablation of the accessory pathway.

Care must be taken in the use of AV nodal blocking agents, especially when the tachycardia is broad complex, since these may paradoxically increase the conduction over the accessory pathway and lead to a 1:1 atrioventricular conduction which may result in ventricular fibrillation.

A simple mnemonic to remember for drugs to avoid in WPW syndrome is ABCD (Adenosine, -Blockers, Calcium Channel Blockers, Digoxin).

👍 👎 Discuss (6) Improve

Next question >

Wolff-Parkinson White ★

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

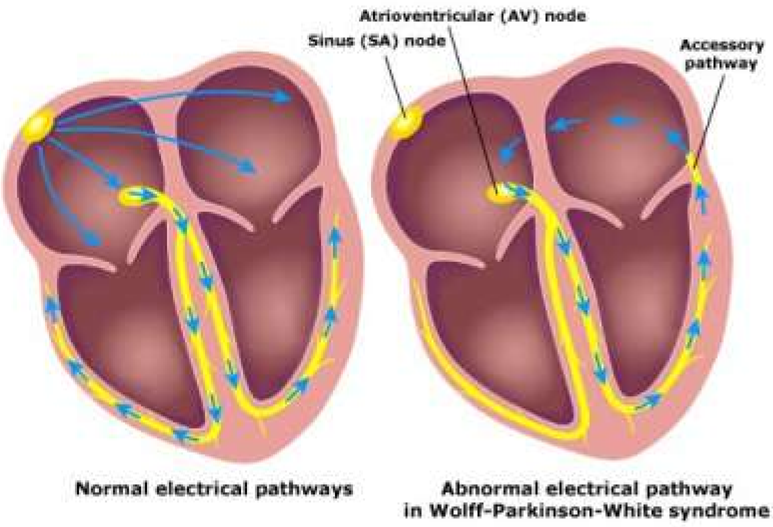
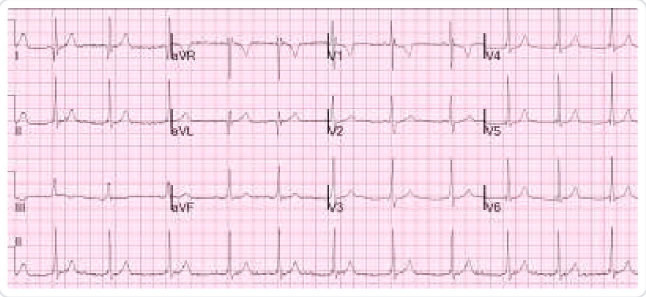

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval

- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
    - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway



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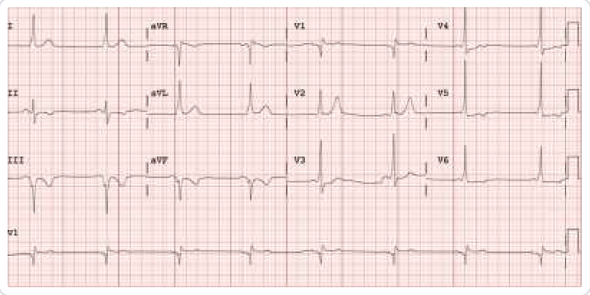

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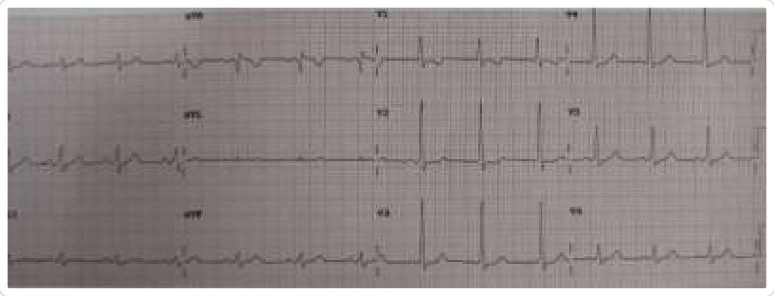

Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
    - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 12 2

Report broken media

Score: **20.6%**

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180 ✗

181 ✗

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A 58-year-old man is admitted with chest pain and shortness of breath. His breathlessness is worsening and he has a new oxygen requirement. His past medical history includes metastatic melanoma, hypertension and diabetes mellitus. He had surgical removal of the original melanoma lesion but now has extensive metastases. He has been seen regularly by the palliative care team as his pain has been very difficult to manage.

His pulse is regular on examination and chest sounds clear on auscultation. His ECG shows sinus rhythm with a rate of 61bpm and QTC is 455ms. His chest x-ray demonstrates clear lung fields. The results of his blood tests are shown below:

Hb	115 g/L	(135-180)
Platelets	451 * 10^9/L	(150 - 400)
WBC	7.2 * 10^9/L	(4.0 - 11.0)
Na^+	142 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Urea	9.6 mmol/L	(2.0 - 7.0)
Creatinine	133 µmol/L	(55 - 120)
CRP	9 mg/L	(< 5)

Which of the following may account for his ECG findings?

○ Digoxin

○ Cyclizine

○ Bisoprolol

○ Amlodipine

○ Methadone

Submit answer

A 58-year-old man is admitted with chest pain and shortness of breath. His breathlessness is worsening and he has a new oxygen requirement. His past medical history includes metastatic melanoma, hypertension and diabetes mellitus. He had surgical removal of the original melanoma lesion but now has extensive metastases. He has been seen regularly by the palliative care team as his pain has been very difficult to manage.

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Urea	9.6 mmol/L	(2.0 - 7.0)
Creatinine	133 µmol/L	(55 - 120)
CRP	9 mg/L	(< 5)

Which of the following may account for his ECG findings?

Digoxin	9%
Cyclizine	19%
Bisoprolol	4%
Amlodipine	1%
Methadone	68%

Methadone is a common cause of QT prolongation

Important for me Less important

This man's ECG shows a prolonged QT interval at 455ms (normal for a male is <430ms).

Methadone is not a drug commonly used but can be used by the palliative care team for patients who are intolerant of opioids and their pain is unmanageable. One of the side effects of methadone is that it prolongs the QT interval and so is the correct answer.

Digoxin is used in the management of atrial fibrillation. Digoxin toxicity is associated with the 'reverse tick' ST morphology change.

Anti-emetics such as ondansetron, not cyclizine are associated with a prolonged QT interval.

Bisoprolol can be associated with bradycardia and atrioventricular block.

Amlodipine may cause palpitations and tachycardia but has no effect on the QT interval.

👍 👎 💬 Discuss (1) Improve

Next question >

Long QT syndrome ★

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
- Jervell-Lange-Nielsen	- amiodarone, sotalol, class 1a	- electrolyte: hypocalcaemia,

Congenital	Drugs*	Other
syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	antiarrhythmic drugs • tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) • methadone • chloroquine • terfenadine** • erythromycin • haloperidol • ondanestron	hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Features

- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management

- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

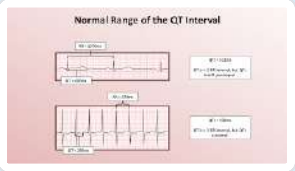
2010 Drug-Induced Long QT syndrome

Life in the Fast Lane 👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link Report broken link

Media



Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

OSMOSIS

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **21%**

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182 ✓

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Question 183 of 328

A 67-year-old male presents with increasing chest pain on exertion over the past 3 weeks, stopping him from going to work. His past medical history includes hypertension, type 2 diabetes mellitus, sick sinus syndrome, recent abstinence from alcohol after a history of excess (with an episode of acute liver decompensation 9 months ago) and stable angina. He previously tried isosorbide mononitrate but reported significant headaches and facial flushing. He has not previously suffered myocardial infarctions, with a recent echo demonstrating 70% ejection fraction. An ECG demonstrates first-degree heart block with normal QRS complexes at 50 beats/ minute, blood pressure is 140/76 mmHg. He currently takes bisoprolol 5mg OD alone and has been using his GTN spray with increasing frequency without effect. What is the most appropriate next step in management?

- ○ Reperfusion therapy with coronary artery bypass graft or percutaneous coronary intervention ×
- ○ Ranolazine ×
- ○ Ivabradine ×
- ○ Diltiazem ×
- ○ Nicorandil ×

Submit answer

Reference ranges

Score: **21%**

1 ×

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182 ✓
183 -

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A 73-year-old man presents to the medical take with a 3-month history of thoracic back pain. Over the last week, he had been having episodes of sweats and shivers, particularly at night.

He was admitted and blood tests were taken.

Haemoglobin	87 g/L
White cells	11.6x10^9/L
Platelets	214 x10^9/L
MCV	70 fl
MCH	20 pg
Blood cultures	*Streptococcus gallolyticus*

MRI showed a discitis at thoracic disks 8/9.
ECHO: No vegetation seen

What is the next investigation for this patient?

- ○ Colonoscopy
- ○ CT chest/abdomen/pelvis
- ○ Ultrasound abdomen/pelvis
- ○ Repeat blood cultures
- ○ PET scan

Submit answer

Reference ranges

Question 184 of 328 ✓

A 73-year-old man presents to the medical take with a 3-month history of thoracic back pain. Over the last week, he had been having episodes of sweats and shivers, particularly at night.

He was admitted and blood tests were taken.

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Platelets	214 x10^9/L
MCV	70 fl
MCH	20 pg
Blood cultures	*Streptococcus gallolyticus*

MRI showed a discitis at thoracic disks 8/9.
ECHO: No vegetation seen

What is the next investigation for this patient?

Colonoscopy	80%
CT chest/abdomen/pelvis	11%
Ultrasound abdomen/pelvis	2%
Repeat blood cultures	4%
PET scan	3%

Streptococcus bovis endocarditis is associated with colorectal cancer

Important for me Less important

Streptococcus gallolyticus is a subtype of *Streptococcus bovis*. *Streptococcus bovis* bacteraemia is associated with underlying colonic malignancies in approximately 10 to 25 percent of patients, therefore, all patients presenting with this should be investigated for rectal cancer. The gold standard investigation is a colonoscopy.

If a malignancy was found on colonoscopy then a CT chest/abdomen/pelvis would be warranted to look for metastases, but it would not be as sensitive as a colonoscopy to look for colonic malignancies. An ultrasound of the abdomen would also be less sensitive to diagnose colonic malignancies than a colonoscopy.

A CT-PET scan may well show a malignancy in the colon, but the patient must be exposed to radioactive dye and radiation, therefore is not the first line investigation.

Repeat blood cultures would be useful when determining the length of treatment needed for this patient, however, they would not aid in the discovery of the underlying diagnosis.

https://academic.oup.com/bmb/article-lookup/doi/10.1093/bmb/ldv027

https://www.britishinfection.org/files/2714/1640/8773/EndocarditisfinalBSAC2012.pdf

👍 👎 Discuss (6) Improve

Next question >

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
    - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
    - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis
    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*
    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link Report broken link

Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

HTTPS://T.ME/+AOWEXXQPZDM1NJZK

BACTERIAL
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **21.3%**

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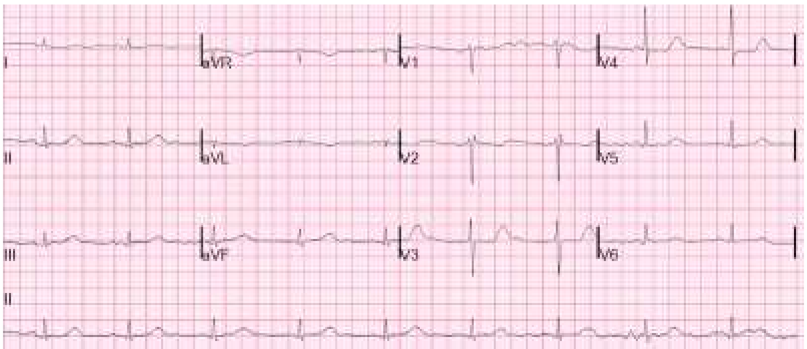
Question 185 of 328

A 26-year-old woman presents to the emergency department with generalised weakness. She has a past medical history of type 1 diabetes mellitus and reports having been vomiting repeatedly over the past 4 days and cannot keep any food or liquids down. There is no history of fever or diarrhoea.

On examination, she appears lethargic with dry mucous membranes. Chest sounds are clear and heart sounds are normal. Her abdomen is soft with generalised tenderness mostly within the epigastric region.

Her blood glucose is recorded as 7.4 mmol/L. An ECG is taken on arrival at the emergency department, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the recommended initial management for this patient?

- ○ Intravenous 0.9% sodium chloride with 40 mmol potassium
- ○ Intravenous 1.26% sodium bicarbonate
- ○ Intravenous calcium gluconate
- ○ Intravenous magnesium sulphate
- ○ Warmed intravenous 0.9% sodium chloride

Submit answer

A 26-year-old woman presents to the emergency department with generalised weakness. She has a past medical history of type 1 diabetes mellitus and reports having been vomiting repeatedly over the past 4 days and cannot keep any food or liquids down. There is no history of fever or diarrhoea.

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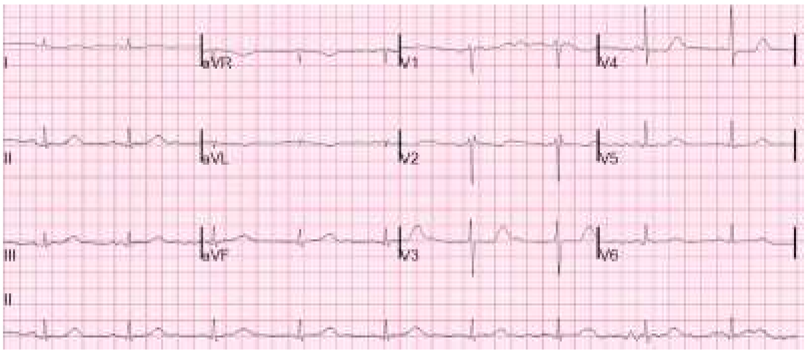


© Image used on license from Dr Smith, University of Minnesota

What is the recommended initial management for this patient?

Intravenous 0.9% sodium chloride with 40 mmol potassium	67%
Intravenous 1.26% sodium bicarbonate	1%
Intravenous calcium gluconate	15%
Intravenous magnesium sulphate	11%
Warmed intravenous 0.9% sodium chloride	6%

This patient's ECG shows U waves that are most noticeable in leads V2 and V3. U waves are small positive deflections that appear shortly after a T wave and frequently represent hypokalaemia. There is also a prolonged

QTc interval (> 440ms). Other features of severe hypokalaemia include increased P wave amplitude, PR interval prolongation and T wave flattening.

Intravenous 0.9% sodium chloride with 40 mmol potassium is correct. There are several causes of U waves including hypothermia, bradycardia, hypocalcaemia, hypomagnesaemia and severe hypokalaemia. There are also multiple causes of a prolonged QTc interval including hypokalaemia, hypomagnesaemia, hypocalcaemia and hypothermia. Given the presentation of prolonged vomiting in a patient with type 1 diabetes (likely representing gastroparesis), this patient is at high risk for having hypokalaemia. The clinical history and ECG are suggestive of hypokalaemia and therefore intravenous potassium replacement is necessary.

Intravenous 1.26% sodium bicarbonate is incorrect. Sodium bicarbonate 1.26% can be administered in instances of metabolic acidosis and certain arrhythmias (eg broad complex tachycardia associated with salicylate overdose). It is not indicated for this patient.

Intravenous calcium gluconate is indicated for severe hypocalcaemia and hyperkalaemia with ECG changes such as tall tented T waves, QRS widening and flattened P waves. Although hypocalcaemia can cause a prolonged QTc interval which is seen here, it is a less common cause of U waves making calcium gluconate a less appropriate drug to administer.

Intravenous magnesium sulphate is given in the management of acute arrhythmias including atrial fibrillation and Torsades de Pointes (a monomorphic ventricular tachycardia associated with QTc prolongation). Although hypomagnesaemia can cause QTc prolongation, it is a less common cause of U waves in comparison to hypokalaemia, making potassium replacement the most appropriate initial management. However, it is important to remember that a normal magnesium level is required for the maintenance of a potassium level within the normal range and, therefore, replacement may also be necessary.

Warmed intravenous 0.9% sodium chloride is incorrect. Hypothermia is a less common cause of U waves and QTc prolongation and more commonly produces J waves which are not seen here. Secondly, there is no indication from the clinical presentation that this patient is hypothermic.

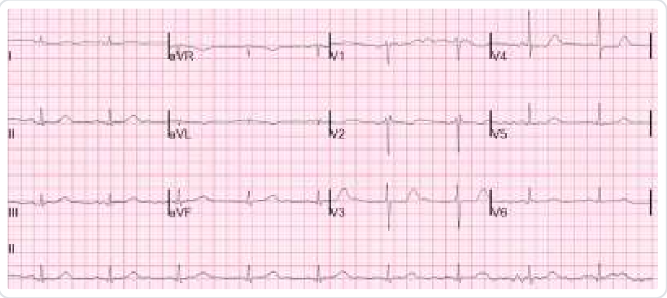
Discuss (2) | Improve

Next question

ECG: hypokalaemia

ECG features of hypokalaemia

- U waves
- small or absent T waves (occasionally inversion)
- prolong PR interval
- ST depression
- long QT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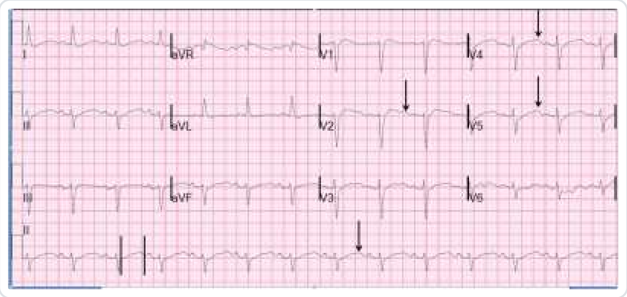
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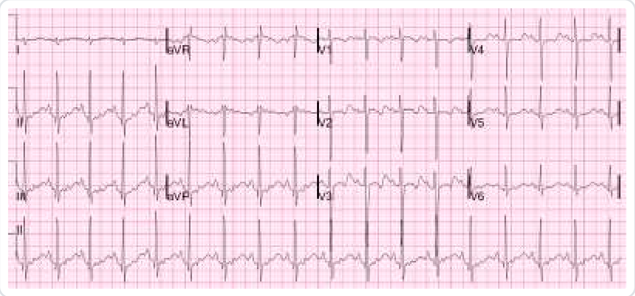
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In Hypokalaemia, U have no Pot and no T, but a long PR and a long QT

+ Q 123

Next question ›

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 48 👎 6

ECG: Hypokalaemia changes

Suggest link Report broken link

Score: **21.7%**

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Question 186 of 328

A 67-year-old gentleman is brought to the resuscitation area of the Emergency Department after an out of hospital cardiac arrest. The paramedics are performing CPR and a laryngeal mask airway in place for ventilation. They inform you that the patient is in asystole and they have already administered IV adrenaline. On the next rhythm check you notice some P-waves on the monitor.

Which of the following is the best management option?

- ○ 300mg IV amiodarone
- ○ IV fluid bolus
- ○ External pacing
- ○ Defibrillation
- ○ 3mg IV atropine

Submit answer

Reference ranges

Score: **21.7%**

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Question 186 of 328

A 67-year-old gentleman is brought to the resuscitation area of the Emergency Department after an out of hospital cardiac arrest. The paramedics are performing CPR and a laryngeal mask airway in place for ventilation. They inform you that the patient is in asystole and they have already administered IV adrenaline. On the next rhythm check you notice some P-waves on the monitor.

Which of the following is the best management option?

300mg IV amiodarone	23%
IV fluid bolus	12%
External pacing	29%
Defibrillation	24%
3mg IV atropine	11%

This patient is in P-wave systole, a special case which does not follow the main cardiac arrest algorithm. In this scenario, external pacing can result in a return of spontaneous circulation.

Atropine is no longer used in any rhythm associated with cardiac arrest, while amiodarone and defibrillation are part of the shockable rhythm protocol. IV fluid bolus is recommended in cardiac arrest where hypovolaemia is suspected as an underlying cause, however, in this scenario, external pacing is more likely to result in the return of spontaneous circulation.

https://www.resus.org.uk/pages/als.pdf

Discuss (8) Improve

Next question >

Adult advanced life support ★

The following is based on the 2021 Resus Council guidelines. Please see the link for more details, below is only a very brief summary of key points.

It should be remembered that the algorithm divides patients into those with:
- 'shockable' rhythms: ventricular fibrillation/pulseless ventricular tachycardia (VF/pulseless VT)
- 'non-shockable' rhythms: asystole/pulseless-electrical activity (asystole/PEA)

Major points include:
- chest compressions
  - the ratio of chest compressions to ventilation is 30:2
  - chest compressions are now continued while a defibrillator is charged
- defibrillation
  - a single shock for VF/pulseless VT followed by 2 minutes of CPR
  - if the cardiac arrested is witnessed in a monitored patient (e.g. in a coronary care unit) then the 2015 guidelines recommend 'up to three quick successive (stacked) shocks', rather than 1 shock followed by CPR
- drug delivery
  - IV access should be attempted and is first-line
  - if IV access cannot be achieved then drugs should be given via the intraosseous route (IO)
  - delivery of drugs via a tracheal tube is no longer recommended
- adrenaline
  - adrenaline 1 mg as soon as possible for non-shockable rhythms
  - during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock
  - repeat adrenaline 1mg every 3-5 minutes whilst ALS continues
- amiodarone
  - amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.
  - a further dose of amiodarone 150 mg should be given to patients who are in VF/pulseless VT after 5 shocks have been administered

    - lidocaine used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead
- thrombolytic drugs
    - should be considered if a pulmonary embolus is suspected
    - if given, CPR should be continued for an extended period of 60-90 minutes

Resuscitation Council UK
GUIDELINES 2021
Adult advanced life support

Other points
- atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA)
- following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

Reversible causes of cardiac arrest:

The 'Hs'	**The 'Ts'**
• Hypoxia • Hypovolaemia • Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders • Hypothermia	• Thrombosis (coronary or pulmonary) • Tension pneumothorax • Tamponade - cardiac • Toxins

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council 👍 32 👎 14

2021 Basic Life Support guidelines

Resuscitation Council 👍 58 👎 25

2021 Advanced Life Support guidelines

Suggest link Report broken link

Media

RC (UK) Cardiac Arrest Management Demo

Resuscitation Council (UK) - YouTube 👍 16 👎 2

Report broken media

Score: **21.6%**

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A 80-year-old man with a past medical history of gout, reflux and ischaemic heart disease is admitted to the emergency department with a atrial fibrillation with fast ventricular response. He is managed according to ALS protocol and is stabilised.

A full set of bloods are sent and are displayed below:

Hb	135 g/l
Platelets	260 * 10^9/l
WBC	6 * 10^9/l

Mg	0.34 µmol/l
Ca (adj)	2.1 u/l
PO4	0.8 u/l

This is discussed with the cardiology registrar, who advises correction of the magnesium.

What medication is the most likely cause of hypomagnesaemia in this case?

- ○ Aspirin ×
- ○ Omeprazole ×
- ○ Ranitidine ×
- ○ Colchicine ×
- ○ Ramipril ×

Submit answer

Reference ranges ⌵

Score: **21.6%**

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Question 187 of 328 ✓

A 80-year-old man with a past medical history of gout, reflux and ischaemic heart disease is admitted to the emergency department with a atrial fibrillation with fast ventricular response. He is managed according to ALS protocol and is stabilised.

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Mg	0.34 µmol/l
Ca (adj)	2.1 u/l
PO4	0.8 u/l

This is discussed with the cardiology registrar, who advises correction of the magnesium.

What medication is the most likely cause of hypomagnesaemia in this case?

Aspirin	1%
Omeprazole	79%
Ranitidine	10%
Colchicine	7%
Ramipril	4%

Careful electrolyte balance is important in the management of arrhythmias. During the generation of the action potential in cardiac pacemaker cells, phase 4 (inflow of potassium) is dependant on magnesium channels, and, although the exact effect in vivo of magnesium administration is unclear, restoring normomagnesaemia is important in patient presenting with dysrhythmias.

PPI use is associated with hypomagnesaemia - the exact mechanism of this is not known, but may be related to poorer intestinal absorption from dietary sources in patients on PPIs.

Ref:
Expert Opinion Drug Saf. 2013 Sep;12(5):709-16. doi: 10.1517/14740338.2013.809062. Epub 2013 Jun 29.
Hypomagnesaemia and proton-pump inhibitors.
Famularo G1, Gasbarrone L, Minisola G.

👍 👎 Discuss (2) Improve

Next question >

Peri-arrest rhythms: tachycardia ★

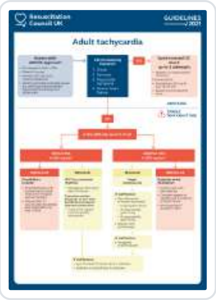
The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given. Up to 3 shocks can be given; after this expert help should be sought.

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment guidelines can be found at the Resuscitation Council website, below is a very limited summary:



Resuscitation Council UK

GUIDELINES 2021

Adult tachycardia

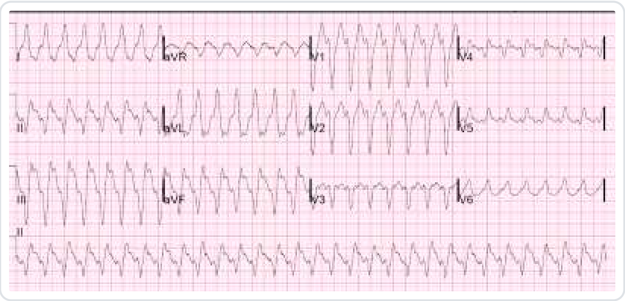
Broad-complex tachycardia

Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion

Irregular

- seek expert help. Possibilities include:
    - atrial fibrillation with bundle branch block - the most likely cause in a stable patient
    - atrial fibrillation with ventricular pre-excitation
    - torsade de pointes



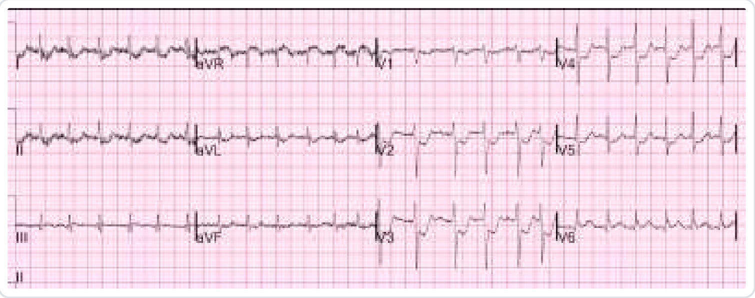
Narrow-complex tachycardia

Regular

- vagal manoeuvres followed by IV adenosine
- if the above is unsuccessful consider a diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control: beta-blockers are usually first-line unless there is a contraindication



123

Next question ›

Textbooks

High-yield textbook

Extended textbook

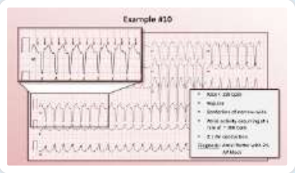
Links

Resuscitation Council UK 30 9

2021 Tachycardia algorithm

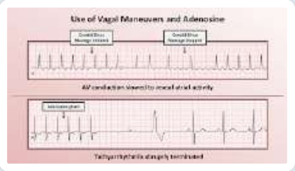
Suggest link Report broken link

Media



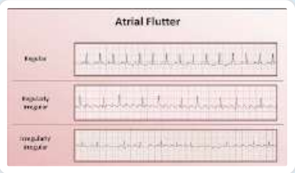
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 5 👎 0


Use of Vagal Maneuvers and Adenosine
AV conduction slowed to reveal atrial activity
Tachyarrhythmia abruptly terminated

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 4 👎 0


Atrial Flutter
Regular
Regularly Irregular
Irregularly Irregular

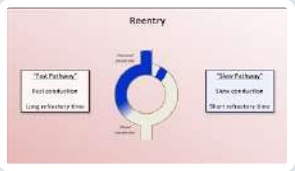
Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 1

CARDIOLOGY
ARRHYTHMIA
TACHYCARDIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 1


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 2 👎 1

Report broken media

Score: **22%**

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Question 188 of 328

A 66-year-old man was admitted from the community with an anterior STEMI to the local district general hospital. He complained of severe chest pain before his wife rang for help. She noted he was clammy and sweaty. About five minutes after being transferred into the emergency department a nurse observed that the patient looked unwell, appeared grey and poorly responsive, . The nurse put out a peri-arrest call.The cardiac monitor showed a broad complex tachycardia. His femoral pulse was faintly palpable and his blood pressure was 76/36 mmHg.

What should be the carried out immediately ?

- ○ Start CPR ×
- ○ IV fluids ×
- ○ DC cardioversion ×
- ○ IV amiodarone ×
- ○ Transfer to tertiary center for immediate PCI ×

Submit answer

Reference ranges

Score: **22%**

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188 -

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Question 188 of 328 ✗

A 66-year-old man was admitted from the community with an anterior STEMI to the local district general hospital. He complained of severe chest pain before his wife rang for help. She noted he was clammy and sweaty. About five minutes after being transferred into the emergency department a nurse observed that the patient looked unwell, appeared grey and poorly responsive, . The nurse put out a peri-arrest call.The cardiac monitor showed a broad complex tachycardia. His femoral pulse was faintly palpable and his blood pressure was 76/36 mmHg.

What should be the carried out immediately ?

Start CPR	7%
IV fluids	2%
DC cardioversion	87%
IV amiodarone	2%
Transfer to tertiary center for immediate PCI	2%

DC cardioversion should be given to a patient presenting with ventricular tachycardia with haemodynamic compromise

Important for me Less important

The answer is immediate DC cardioversion for a patient with ventricular tachycardia presenting with haemodynamic compromise. If the patient was stable, amiodarone and magnesium could be considered. It is important to check the electrolytes. Ideally, the patient needs PCI but must be stable before transfer can take place. There is no role for CPR as this is not a cardiac arrest situation.

Discuss (3) Improve

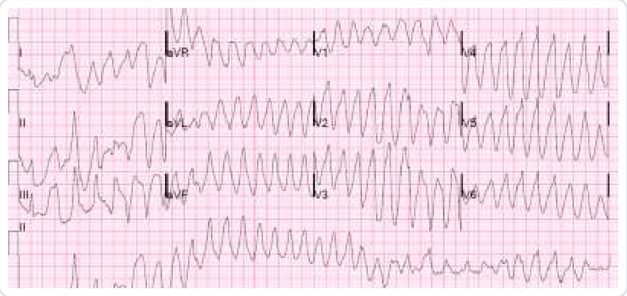
Next question ›

Ventricular tachycardia

Ventricular tachycardia (VT) is broad-complex tachycardia originating from a ventricular ectopic focus. It has the potential to precipitate ventricular fibrillation and hence requires urgent treatment.

There are two main types of VT:

- monomorphic VT: most commonly caused by myocardial infarction
- polymorphic VT: A subtype of polymorphic VT is torsades de pointes which is precipitated by prolongation of the QT interval. The causes of a long QT interval are listed below



Causes of a prolonged QT interval

Congenital	Drugs	Other
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, fluoxetine • chloroquine • terfenadine • erythromycin	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Management

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy
- amiodarone: ideally administered through a central line
- lidocaine: use with caution in severe left ventricular impairment
- procainamide

Verapamil should NOT be used in VT

If drug therapy fails
- electrophysiological study (EPS)
- implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 0 👎 0

2021 Tachycardia algorithm

Suggest link Report broken link

Media

Ventricular tachycardia

Osmosis - YouTube 👍 2 👎 0

Ventricular Tachycardia (V-tach)
Khan Academy

Ventricular tachycardia

Khan Academy - YouTube 👍 1 👎 0

Report broken media

Score: **21.9%**

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K^+	5.5 mmol/L	(3.5 - 5.0)
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Creatinine	188 µmol/L	(55 - 120)

The echocardiogram shows:

Summary: Left ventricular systolic function is moderately impaired. The ejection fraction is visually estimated at ~40%.

Regular furosemide is started.

What additional medication should be prescribed?

○ Amlodipine

○ Dapagliflozin

○ Ivabradine ×

○ Sacubitril and valsartan ×

○ Spironolactone ×

Submit answer

Reference ranges ∨

Score: **21.9%**

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Summary: Left ventricular systolic function is moderately impaired. The ejection fraction is visually estimated at ~40%.

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What additional medication should be prescribed?

Amlodipine	5%
Dapagliflozin	53%

Ivabradine	6%
Sacubitril and valsartan	15%
Spironolactone	21%

SGLT-2 inhibitors should be considered as second-line therapy in patients with HFrEF in addition to optimised standard care

Important for me Less important

Dapagliflozin is correct. This patient has evidence of heart failure with reduced ejection fraction on his echocardiogram. The first-line treatment would be an ACE inhibitor and a beta blocker, but this patient already takes both of these agents. The second-line treatment is usually an aldosterone antagonist such as spironolactone, which can then be complemented with an SGLT2 inhibitor such as dapagliflozin to reduce mortality further. Given that this patient is already hyperkalaemic, spironolactone would be an inappropriate choice, and an SGLT2 inhibitor would be preferable.

Amlodipine is incorrect. Amlodipine would be indicated if the patient required an additional antihypertensive agent, but in this case, the blood pressure is within acceptable parameters, and amlodipine is not otherwise indicated in the management of heart failure. It may even worsen symptoms due to its vasodilatory effect, leading to fluid retention.

Ivabradine is incorrect. Ivabradine is an inhibitor of the funny current in cardiac pacemaker cells. It is used in the management of angina and heart failure. In heart failure, however, it is only recommended as third-line therapy and only in those in sinus rhythm with a ventricular rate over 75 bpm AND an ejection fraction of less than 35%. If ivabradine is used in patients with a resting heart rate below 75 bpm, the risk of bradycardia increases significantly. This patient is not yet on second-line therapy and meets neither of the above criteria, so this answer is incorrect.

Sacubitril and valsartan is incorrect. The combination of sacubitril and valsartan is recommended as a third-line therapy in the management of chronic heart failure, but only in those with a left ventricular fraction of less than 35%. This combination requires initiation by a specialist after an

ACE inhibitor or ARB wash-out period. This patient has not yet been tried on second-line therapy; however, aldosterone antagonists +/—SGLT2 inhibitors would be considered first.

Spironolactone is incorrect. Second-line therapy in the management of stable heart failure is an aldosterone antagonist such as spironolactone. SGLT2 inhibitors may also be added at this stage. Given that this patient is already hyperkalaemic, an aldosterone antagonist would be contraindicated, making an SGLT2 inhibitor the correct answer in this case.

👍 👎 Discuss (3) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction

- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

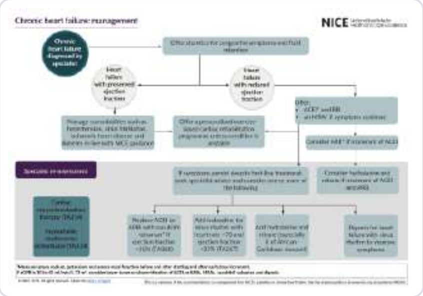
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period
- digoxin
  - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
  - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
  - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
  - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

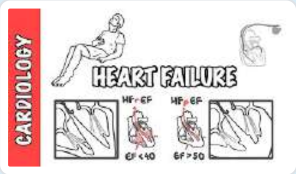
2012 Guidance on ivabradine

NICE 42 15

2018 Chronic heart failure guidelines

Suggest link Report broken link

Media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HEART FAILURE
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HFpEF
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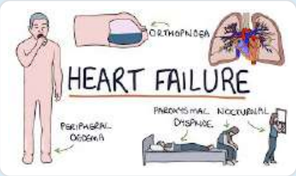
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1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Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **21.8%**

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A 52-year-old gentleman works as a lorry driver. His past medical history includes hypertension and raised BMI. He is admitted to the Emergency Department with chest pain. An ECG confirms ST depression in V1 to V4, with no pathological Q waves. Subsequent blood tests reveal a troponin of 10,000 consistent with a non-ST-elevation myocardial infarction (NSTEMI). He is commenced on appropriate secondary prevention, including aspirin 75mg OD, clopidogrel 75mg OD and fondaparinux 2.5mg SC. The patient is transferred to the cardiology unit and awaits in-patient angiography.

You are asked to review him urgently as he describes acute breathlessness. He denies chest pain. Observations are as follows: heart rate 130 beats per minute, blood pressure 95/62 mmHg, temperature 36.1ºC, respiratory rate 28/min, saturations 94% on 10 litres oxygen. On examination, heart sounds are normal with no added sounds. JVP is mildly elevated. There is no evidence of ascites or peripheral oedema. A repeat ECG confirms sinus tachycardia (heart rate 130bpm) but no dynamic changes. A portable chest x-ray reveals cardiomegaly with upper lobe diversion and peri-hilar shadowing. An urgent bedside echocardiogram demonstrates significant LV systolic dysfunction with a 2cm of pericardial effusion but no features of tamponade.

What is the single next best management step?

○ Continuous positive airway pressure (CPAP)

○ Ultrasound-guided pericardiocentesis

○ Urgent angiography

○ IV fluids

○ Inotrope and IV diuretic

Submit answer

Reference ranges

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What is the single next best management step?

Continuous positive airway pressure (CPAP)	20%
Ultrasound-guided pericardiocentesis	25%
Urgent angiography	16%
IV fluids	3%
Inotrope and IV diuretic	36%

This patient is demonstrating features of cardiogenic shock, secondary to anterior wall myocardial infarction. With anterior infarcts, the mainstay of treatment includes diuretics to reduce pulmonary congestion and inotropic support to enhance cardiac output and perfusion.

Echocardiography demonstrates a reasonable-sized pericardial effusion but there is no indication for pericardiocentesis in the absence of features consistent with cardiac tamponade. Intravenous fluids would exacerbate pulmonary oedema in the context of anterior infarcts and are relatively contraindicated for this reason. They are of relevance, however, in inferior infarcts where preload needs to be maintained. This patient is preserving oxygen saturations via Venturi mask and therefore, CPAP is not currently indicated. Angiography is required to clarify the extent of coronary disease in due course, but the patient requires stabilisation before this can occur.

👍 | 👎 | Discuss (13) | Improve

Next question >

Myocardial infarction: complications

Patients are at risk of a number of immediate, early and late complications following a myocardial infarction (MI).

Cardiac arrest

This most commonly occurs due to patients developing ventricular fibrillation and is the most common cause of death following a MI. Patients are managed as per the ALS protocol with defibrillation.

Cardiogenic shock

If a large part of the ventricular myocardium is damaged in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock. This is difficult to treat. Other causes of cardiogenic shock include the 'mechanical' complications such as left ventricular free wall rupture as listed below. Patients may require inotropic support and/or an intra-aortic balloon pump.

Chronic heart failure

As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop diuretics such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias

Ventricular fibrillation, as mentioned above, is the most common cause of death following a MI. Other common arrhythmias including ventricular tachycardia.

Bradyarrhythmias

Atrioventricular block is more common following inferior myocardial infarctions.

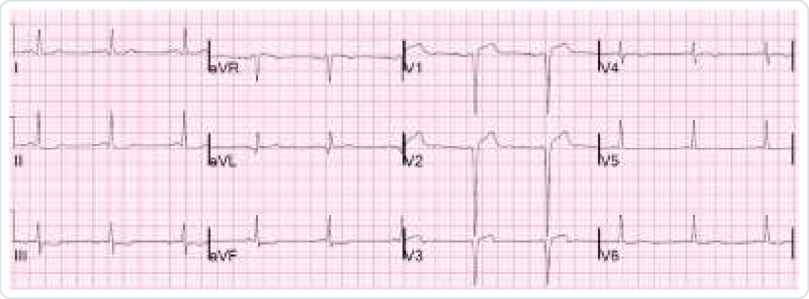
Pericarditis

Pericarditis in the first 48 hours following a transmural MI is common (c. 10% of patients). The pain is typical for pericarditis (worse on lying flat etc), a pericardial rub may be heard and a pericardial effusion may be demonstrated with an echocardiogram.

Dressler's syndrome tends to occur around 2-6 weeks following a MI. The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers. It is characterised by a combination of fever, pleuritic pain, pericardial effusion and a raised ESR. It is treated with NSAIDs.

Left ventricular aneurysm

The ischaemic damage sustained may weaken the myocardium resulting in aneurysm formation. This is typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated.



Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent pericardiocentesis and thoracotomy are required.

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. Acute hypotension and pulmonary oedema may occur. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 36 👎 22

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Suggest link Report broken link

Score: **22.2%**

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Question 191 of 328

A 35-year-old man presents to the emergency department with palpitations that came on suddenly earlier that afternoon. He has had symptoms like this on 3 previous occasions which resolved by themselves after a few minutes. He smokes a pack of cigarettes a day and drinks 5-6 pints of beer during the weekends.

He works as a builder on a construction site. He has no significant medical conditions and does not take any regular medication. In the emergency department, his heart rate is 180 beats per minute and his blood pressure 134/62 mmHg. He has no shortness of breath or chest pain but continues to have significant palpitations. His ECG shows a rapid regular rhythm with a QRS interval of 89 ms. On physical examination, he is comfortable at rest, and peripherally warm and well perfused. His JVP is not visible, and heart sounds are normal without any added sounds or murmurs, his chest is clear to auscultation, his abdomen is soft and non-tender, and he has no swelling of his lower limbs. He is attached to a cardiac monitor and is asked to blow forcefully into a syringe on 2 separate occasions which does not improve his symptoms.

What is the next step in management?

○ Perform carotid sinus massage at the bedside

○ Administer 5 mg of IV verapamil

○ Administer 6 mg of IV adenosine

○ Administer 12 mg of IV adenosine

○ Administer sedation and prepare for direct current cardioversion

Submit answer

Reference ranges

Score: **22.2%**

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Question 191 of 328 ✓

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What is the next step in management?

Perform carotid sinus massage at the bedside	25%
Administer 5 mg of IV verapamil	1%
[✓ Administer 6 mg of IV adenosine]	72%
Administer 12 mg of IV adenosine	1%
Administer sedation and prepare for direct current cardioversion	0%

Patients with SVT who are haemodynamically stable and who do not respond to vagal manoeuvres, the next step is treating with adenosine

Important for me Less important

This young man presents with a likely supraventricular tachycardia. He has symptoms with palpitations, but does not have hemodynamic compromise, nor does he have signs or symptoms of cardiac ischemia or acute heart failure. He, therefore, does not need to be managed with direct current cardioversion. He has already tried vagal manoeuvres, by blowing forcefully into a syringe. The next step in his management is administrating a bolus of IV adenosine at a dose of 6 mg initially, in an attempt to slow down his heart rate and reveal the underlying rhythm.

He has already tried vagal manoeuvres on 2 occasions unsuccessfully and it would be inappropriate to try vagal manoeuvres again while forgoing giving adenosine which would be the next step in his management.

If the initial dose of 6 mg of IV adenosine is unsuccessful then he can be trialled with further adenosine at a higher dose of 12 mg. If these higher doses of adenosine are unsuccessful and he remains hemodynamically stable he could be treated with IV verapamil.

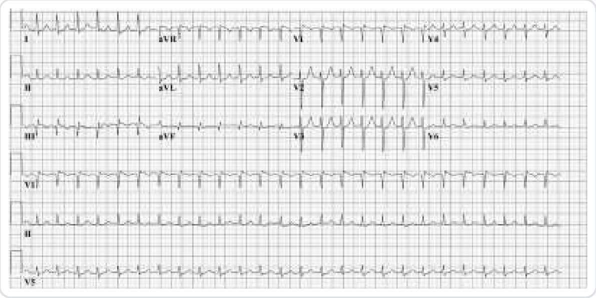
This patient is hemodynamically stable and does not have clinical features to suggest cardiac ischemia or acute heart failure and so does not require direct current cardioversion at present.

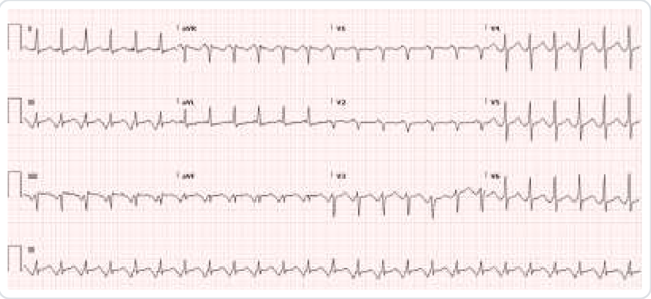
👍 👎 💬 Discuss (4) Improve

Next question ›

Supraventricular tachycardia ★

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
    - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
    - carotid sinus massage
- intravenous adenosine
    - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
    - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

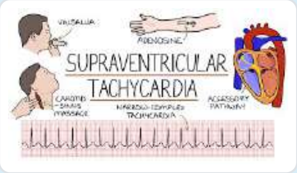
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

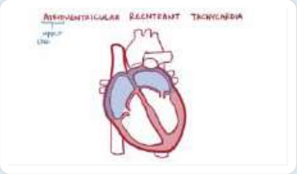
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

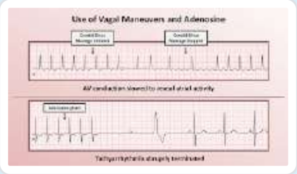
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0


Use of Vagal Maneuvers and Adenosine
Tachyarrhythmia abruptly terminated

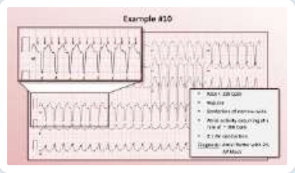
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0

Atrioventricular nodal re-entrant tachycardia (AVNRT)
AVRT & AVNRT
Khan Academy

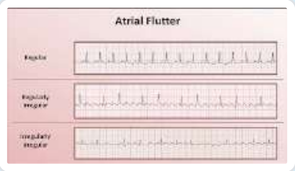
Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0



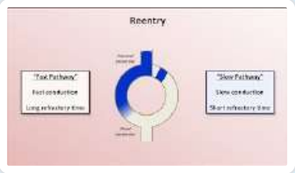
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0


Atrial Flutter
Regular
Regularly irregular
Irregularly irregular

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **22.6%**

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A 13 year-old girl presents with chest pain and fever.

On examination the temperature is 38.1ºC, heart rate is 90 beats/minute and respiratory rate is 18 breaths/minute. The chest is clear to auscultation. There is an early diastolic murmur at the left sternal edge.

ECG reveals sinus rhythm, PR interval 210ms.

Blood tests reveal:

Hb	121 g/l
Platelets	420 * 10^9/l
WBC	9.3 * 10^9/l
Na^+	136 mmol/l
K^+	3.7 mmol/l
Urea	3.8 mmol/l
Creatinine	72 µmol/l
Bilirubin	22 µmol/l
ALP	110 u/l
ALT	53 u/l
Albumin	36 g/l
C-reactive protein	36
Antistreptolysin O antibody titre	320 units/ml

Which other sign may also be present in this patient?

- ○ Dermatitis herpetiformis
- ○ Erythema marginatum
- ○ Pyoderma gangrenosum
- ○ Vitiligo

○ Livedo reticularis ×

Submit answer

Reference ranges ∨

Score: **22.6%**

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On examination the temperature is 38.1ºC, heart rate is 90 beats/minute and respiratory rate is 18 breaths/minute. The chest is clear to auscultation. There is an early diastolic murmur at the left sternal edge.

ECG reveals sinus rhythm, PR interval 210ms.

Blood tests reveal:

Hb	121 g/l
Platelets	420 * 10^9/l
WBC	9.3 * 10^9/l
Na^+	136 mmol/l
K^+	3.7 mmol/l
Urea	3.8 mmol/l
Creatinine	72 µmol/l
Bilirubin	22 µmol/l
ALP	110 u/l
ALT	53 u/l
Albumin	36 g/l
C-reactive protein	36
Antistreptolysin O antibody titre	320 units/ml

Which other sign may also be present in this patient?

Dermatitis herpetiformis	2%
Erythema marginatum	84%
Pyoderma gangrenosum	3%
Vitiligo	1%

Livedo reticularis 10%

Acute rheumatic fever (ARF) is caused by a reaction to a bacterial infection with particular strains of group A streptococcus.

Symptoms of ARF generally develop several weeks after an episode of streptococcal pharyngitis

ARF causes a variety of characteristic clinical features:
- Polyarthritis
- Carditis
- Sydenham's chorea
- Erythema marginatum (A characteristic skin rash that occurs in about 10% of first attacks of ARF in children. it is rare in adults). The rash appears as pink or red macules (flat spots) or papules (small lumps), which spread outwards in a circular shape. As the lesions advance, the edges become raised and red, and the centre clears.

Source: http://www.dermnetnz.org/bacterial/rheumatic-fever.html

👍 👎 Discuss (4) Improve

Next question >

Rheumatic fever ★

Rheumatic fever develops following an immunological reaction to a recent (2-4 weeks ago) *Streptococcus pyogenes* infection.

Pathogenesis
- *Streptococcus pyogenes* infection → activation of the innate immune system leading to antigen presentation to T cells
- B and T cells produce IgG and IgM antibodies and CD4+ T cells are activated
- there is then a cross-reactive immune response (a form of type II hypersensitivity) thought to be mediated by molecular mimicry
- the cell wall of *Streptococcus pyogenes* includes M protein, a virulence factor that is highly antigenic. It is thought that the

antibodies against M protein cross-react with myosin and the smooth muscle of arteries
- this response leads to the clinical features of rheumatic fever
- Aschoff bodies describes the granulomatous nodules found in rheumatic heart fever

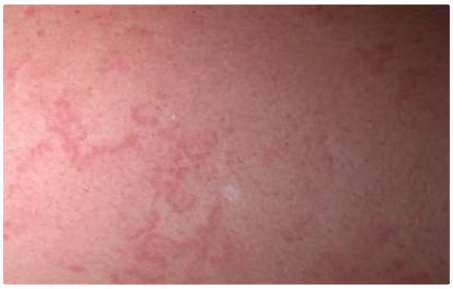
Diagnostic criteria

Diagnosis is based on evidence of recent streptococcal infection accompanied by:
- 2 major criteria
- 1 major with 2 minor criteria

Evidence of recent streptococcal infection
- raised or rising streptococci antibodies,
- positive throat swab
- positive rapid group A streptococcal antigen test

Major criteria
- erythema marginatum
- Sydenham's chorea: this is often a late feature
- polyarthritis
- carditis and valvulitis (eg, pancarditis)
    - The latest iteration of the Jones criteria (published in 2015) state that rheumatic carditis cannot be based on pericarditis or myocarditis alone and that there must be evidence of endocarditis (the clinical correlate of which is valvulitis which manifests as a regurgitant murmur)
- subcutaneous nodules

Minor criteria
- raised ESR or CRP
- pyrexia
- arthralgia (not if arthritis a major criteria)
- prolonged PR interval



Management

Outline of management

- antibiotics: oral penicillin V
- anti-inflammatories: NSAIDs are first-line
- treatment of any complications that develop e.g. heart failure

＋ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ 👍 8 👎 5

Rheumatic fever

Suggest link Report broken link

Media

RHEUMATIC FEVER
&
HEART DISEASE
Osmosis.org

Rheumatic fever & heart disease

Osmosis - YouTube 👍 6 👎 0

Report broken media

Score: **22.5%**

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← Question 193 of 328 →

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- ○ Inferior non-ST elevation myocardial infarction (NSTEMI) ×
- ○ Hypertrophic obstructive cardiomyopathy ×
- ○ Prinzmetal angina ×
- ○ Pulmonary embolus ×
- ○ Takotsubo cardiomyopathy ×

Submit answer

Reference ranges ∨

Score: **22.5%**

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193 -

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Inferior non-ST elevation myocardial infarction (NSTEMI)	1%
Hypertrophic obstructive cardiomyopathy	4%
Prinzmetal angina	7%
Pulmonary embolus	1%
Takotsubo cardiomyopathy	86%

This is a patient with cardiac sounding chest pain with a troponin positive event, in the absence of vascular risk factors, and in the context of normal coronary vessels. The diagnosis is made on the LV description: apical ballooning is pathognomonic of Takotsubo cardiomyopathy, with the shape of the heart named after the Japanese octopus trap. It classically presents in post-menopausal women and is particularly triggered by anxiety and emotional stress. Treatment is supportive as per heart failure, possibly requiring inotropes if there is no LV outflow tract obstruction and with beta blockade, if there is LV outflow tract obstruction. Patients generally recover their LV ejection fraction within 4 weeks.

Discuss (1) Improve

Next question

Takotsubo cardiomyopathy

Takotsubo cardiomyopathy is a type of non-ischaemic cardiomyopathy associated with a transient, apical ballooning of the myocardium. It may be triggered by stress.

Pathophysiology

- Takotsubo is a Japanese word that describes an octopus trap
- the apical ballooning appearance occurs due to severe hypokinesis of the mid and apical segments with preservation of activity of the basal segments. In simple terms, the bottom of the heart (the apex) does not contract and therefore appears to balloon out. However, the area closer to the top (the base) continues to contract (creating the neck of the octopus trap)

Features

- chest pain
- features of heart failure
- ECG: ST-elevation
- normal coronary angiogram

Treatment is supportive.

Prognosis

- the majority of patients improve with supportive treatment

+ Q 123

Next question

Textbooks

High-yield textbook

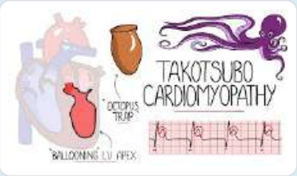
Extended textbook

Media

CARDIOLOGY
TAKOTSUBO CARDIOMYOPATHY

Takotsubo cardiomyopathy

Armando Hasudungan - YouTube 👍 4 👎 0


TAKOTSUBO CARDIOMYOPATHY
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BALLOONING LV APEX

Takotsubo cardiomyopathy

Rhesus Medicine - YouTube 👍 0 👎 0

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Question 194 of 328

A 69-year-old gentleman presents to the emergency department with shortness of breath. He has been experiencing worsening palpitations since he was diagnosed with a chest infection two days ago by his GP and started on oral antibiotics. He is now feeling very unwell and severely short of breath. He has a past medical history of myocardial infarction, AF and type 2 diabetes mellitus. On examination he has a heart rate of 167bpm, blood pressure of 87/50mmHg, oxygen saturations of 93% on 5 litres of oxygen and a temperature of 36.5ºC. He appears very unwell and distressed with bilateral crepitations to his mid-zones and peripheral oedema to his thighs. His ECG shows atrial fibrillations. What is the most appropriate immediate management?

○ IV fluids ×

○ Synchronised DC cardioversion ×

○ Unsynchronised DC cardioversion ×

○ IV amiodarone ×

○ IV adenosine ×

Submit answer

Reference ranges

Score: **22.4%**

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IV fluids	3%
Synchronised DC cardioversion	86%
Unsynchronised DC cardioversion	8%
IV amiodarone	3%
IV adenosine	0%

In AF with decompensation the correct management is synchronised DC cardioversion

Important for me Less important

The patient should have a synchronised DC cardioversion attempted immediately. This patient has a tachyarrhythmia which has clearly decompensated him with evidence of acute heart failure and shock. This may have occurred due to underlying infection. The shock must be synchronised to deliver it during an R wave. This means that the defibrillator will detect R waves and when the shock is administered the machine will pause until it identifies an R wave and then deliver the shock. The purpose of this is primarily to avoid shocking during a T wave which could cause asystole. A shock without synchronisation is used in the resuscitation algorithm during VF and pulseless VT. Amiodarone should be used if three shocks fail to restore rhythm and adenosine is used in haemodynamically stable narrow complex tachycardia. Fluids may worsen pulmonary oedema in this setting.

👍 👎 Discuss (8) Improve

Next question >

Atrial fibrillation: cardioversion ★

There are two scenarios where cardioversion may be used in atrial fibrillation:

- electrical cardioversion as an emergency if the patient is haemodynamically unstable
- electrical or pharmacological cardioversion as an elective procedure where a rhythm control strategy is preferred.

Electrical cardioversion is synchronised to the R wave to prevent delivery of a shock during the vulnerable period of cardiac repolarisation when ventricular fibrillation can be induced.

The notes below refer to cardioversion being used in the elective scenario for rhythm control. The wording of the 2014 NICE guidelines is as follows:

> *offer rate or rhythm control if the onset of the arrhythmia is less than 48 hours, and start rate control if it is more than 48 hours or is uncertain*

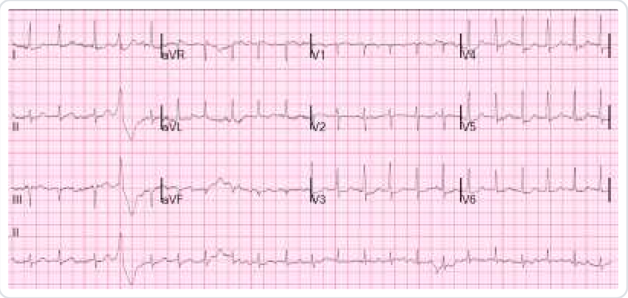
Onset < 48 hours

If the atrial fibrillation (AF) is definitely of less than 48 hours onset patients should be heparinised. Patients who have risk factors for

ischaemic stroke should be put on lifelong oral anticoagulation.
Otherwise, patients may be cardioverted using either:

- electrical - 'DC cardioversion'
- pharmacology - amiodarone if structural heart disease, flecainide or amiodarone in those without structural heart disease

Following electrical cardioversion if AF is confirmed as being less than 48 hours duration then further anticoagulation is unnecessary



Onset > 48 hours

If the patient has been in AF for more than 48 hours then anticoagulation should be given for at least 3 weeks prior to cardioversion. An alternative strategy is to perform a transoesophageal echo (TOE) to exclude a left atrial appendage (LAA) thrombus. If excluded patients may be heparinised and cardioverted immediately.

NICE recommend electrical cardioversion in this scenario, rather than pharmacological.

If there is a high risk of cardioversion failure (e.g. Previous failure or AF recurrence) then it is recommend to have at least 4 weeks amiodarone or sotalol prior to electrical cardioversion

Following electrical cardioversion patients should be anticoagulated for at least 4 weeks. After this time decisions about anticoagulation should be taken on an individual basis depending on the risk of recurrence

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 22 👎 9

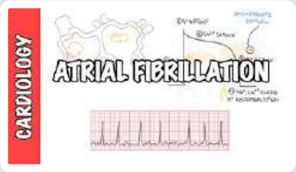
2021 Atrial fibrillation guidelines

European Society of Cardiology 👍 15 👎 3

2020 Atrial fibrillation guidelines

Suggest link Report broken link

Media



Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 1 👎 0

Atrial fibrillation

Osmosis - YouTube 👍 1 👎 0

Report broken media

Score: **22.3%**

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A 53-year-old woman is found to have a systolic murmur on a routine check-up. A transthoracic echocardiogram reveals severe primary mitral regurgitation and a left ventricular ejection fraction of 55%, with other valves showing no significant pathology. A routine ECG demonstrates sinus rhythm. On questioning, she has no attributable symptoms such as shortness of breath, reduced exercise tolerance or chest pain. She is normally very active and works in an insurance firm. She has a past medical history of well-controlled asthma, post-partum depression and a tibial fracture one year ago. She has a salbutamol inhaler but no other regular medications. What is the most appropriate management plan?

○ Follow-up in six months ×

○ Start nitrites ×

○ Stop salbutamol ×

○ Exercise testing ×

○ Referral for mitral valve surgery ×

Submit answer

Reference ranges ⌄

Score: **22.3%**

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A 53-year-old woman is found to have a systolic murmur on a routine check-up. A transthoracic echocardiogram reveals severe primary mitral regurgitation and a left ventricular ejection fraction of 55%, with other valves showing no significant pathology. A routine ECG demonstrates sinus rhythm. On questioning, she has no attributable symptoms such as shortness of breath, reduced exercise tolerance or chest pain. She is normally very active and works in an insurance firm. She has a past medical history of well-controlled asthma, post-partum depression and a tibial fracture one year ago. She has a salbutamol inhaler but no other regular medications. What is the most appropriate management plan?

Follow-up in six months	61%
Start nitrites	1%
Stop salbutamol	1%
Exercise testing	6%
Referral for mitral valve surgery	31%

The correct answer is a referral for mitral valve surgery. This asymptomatic patient with mitral regurgitation who has an ejection fraction of 55%. As this is below 60% the patient should be referred for surgery. Other surgical indications include new onset of AF or high likelihood of durable repair with low surgical risk and absence of risk factors. Follow-up in six months would be appropriate if there were no indications for surgery and medical management, such as nitrites, would be indicated if the patient was symptomatic and at too high risk for surgery. Stopping the salbutamol is unlikely to make any significant difference.

Source:
Vahanian A., Alfieri O., Andreotti F., et al. Guidelines on the management of valvular heart disease (version 2012). The Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery.

European Heart Journal. 2012;33:24512496. doi: 10.1093/eurheartj/ehs109

👍 👎 Discuss (9) Improve

Next question >

Mitral regurgitation ★

Also known as mitral insufficiency, mitral regurgitation (MR) occurs when blood leaks back through the mitral valve on systole. It is the second most common valve disease after aortic stenosis. The mitral valve is located between the left atrium and ventricle, and regurgitation leads to a less efficient heart as less blood is pumped through the body with each contraction. This said, MR is common in otherwise healthy patients to a trivial degree and does not need treatment.

As the degree of regurgitation becomes more severe, the body's oxygen demands may exceed what the heart can supply and as a result, the myocardium can thicken over time. While this may be benign initially, patients may find themselves increasingly fatigued as a thicker myocardium becomes less efficient, and eventually go into irreversible heart failure.

Risk factors

- Female sex
- Lower body mass
- Age
- Renal dysfunction
- Prior myocardial infarction
- Prior mitral stenosis or valve prolapse
- Collagen disorders e.g. Marfan's Syndrome and Ehlers-Danlos syndrome

Causes

- Following coronary artery disease or post-MI: if the papillary muscles or chordae tendinae are affected by a cardiac insult, mitral valve disease may ensue as a result of damage to its supporting structures.

- Mitral valve prolapse: Occurs when the leaflets of the mitral valve is deformed so the valve does not close properly and allows for backflow. Most patients with this have a trivial degree of mitral regurgitation.
- Infective endocarditis: When vegetations from the organisms colonising the heart grow on the mitral valve, it is prevented from closing properly. Patients with abnormal valves are more likely to develop endocarditis as opposed to their peers.
- Rheumatic fever: While this is uncommon in developed countries, rheumatic fever can cause inflammation of the valves and therefore result in mitral regurgitation.
- Congenital

Symptoms

- Most patients with MR are asymptomatic, and patients suffering from mild to moderate MR may stay largely asymptomatic indefinitely. Symptoms tend to be due to failure of the left ventricle, arrhythmias or pulmonary hypertension. This may present as fatigue, shortness of breath and oedema.

Signs

- The murmur heard on auscultation of the chest is typically a pansystolic murmur described as 'blowing'. It is heard best at the apex and radiating into the axilla. S1 may be quiet as a result of incomplete closure of the valve. Severe MR may cause a widely split S2

Investigations

- ECG may show a broad P wave, indicative of atrial enlargement
- Cardiomegaly may be seen on chest x-ray, with an enlarged left atrium and ventricle
- Echocardiography is crucial to diagnosis and to assess severity

Treatment options

- Medical management in acute cases involves nitrates, diuretics, positive inotropes and an intra-aortic balloon pump to increase cardiac output
- If patients are in heart failure, ACE inhibitors may be considered along with beta-blockers and spironolactone
- In acute, severe regurgitation, surgery is indicated

- The evidence for repair over replacement is strong in degenerative regurgitation, and is demonstrated through lower mortality and higher survival rates
- When this is not possible, valve replacement with either an artificial valve or a pig valve is considered

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Next question >

Textbooks

High-yield textbook

Extended textbook

Media

MITRAL REGURGITATION (INSUFFICIENCY)

Mitral regurgitation

Armando Hasudungan - YouTube 5 0

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 1 0

Mitral regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1

Report broken media

Score: **22.2%**

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A 55-year-old man with type 2 diabetes comes to the Emergency department with symptoms of worsening cardiac failure. Current medication includes metformin and empagliflozin for blood glucose control, ramipril, doxazosin, furosemide, aspirin and atorvastatin. His blood pressure is 112/70 mmHg, his pulse is 80 beats per minute and regular. There are bilateral crackles to the mid zones on auscultation of the chest, and he has pitting oedema to the mid-shins bilaterally.

Investigations

Na^+	138 mmol/l
K^+	4.5 mmol/l
Urea	6.2 mmol/l
Creatinine	112 µmol/l

Which of the following medications would you discontinue?

- ○ Atorvastatin ×
- ○ Doxazosin ×
- ○ Empagliflozin ×
- ○ Metformin ×
- ○ Ramipril ×

Submit answer

Reference ranges

Score: **22.2%**

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A 55-year-old man with type 2 diabetes comes to the Emergency department with symptoms of worsening cardiac failure. Current medication includes metformin and empagliflozin for blood glucose control, ramipril, doxazosin, furosemide, aspirin and atorvastatin. His blood pressure is 112/70 mmHg, his pulse is 80 beats per minute and regular. There are bilateral crackles to the mid zones on auscultation of the chest, and he has pitting oedema to the mid-shins bilaterally.

Investigations

Na^+	138 mmol/l
K^+	4.5 mmol/l
Urea	6.2 mmol/l
Creatinine	112 µmol/l

Which of the following medications would you discontinue?

Atorvastatin	1%
Doxazosin	50%
Empagliflozin	15%
Metformin	24%
Ramipril	10%

In this situation of worsening heart failure, discontinuation of the alpha-blocker doxazosin may well improve symptoms. The ALLHAT trial suggested that alpha blockade versus other antihypertensive options was associated with a 25% greater risk of congestive cardiac failure. Guidelines recommend that heart failure is best managed with ACE inhibition, a cardioselective beta blocker rather than an alpha blocker, and loop diuresis if required for fluid overload.

https://www.nice.org.uk/guidance/Cg108

Although atorvastatin may be associated with myositis, it isn't thought to exacerbate heart failure. Evidence for empagliflozin, as one of the SGLT2 inhibitor class, is that it promotes sodium excretion and has a thiazide diuretic like effect with some benefit in patients with incipient cardiac failure. Metformin has no negative impact on heart failure. Metformin is only really contraindicated in a period of acute hypotension, not the chronic heart failure setting seen here.

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Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**

- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone

- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

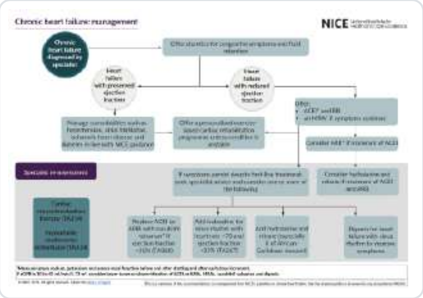
There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments
- offer **annual influenza vaccine**

- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 14 12

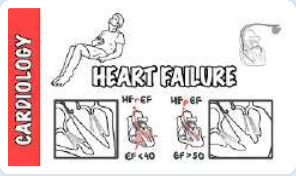
2012 Guidance on ivabradine

NICE 42 15

2018 Chronic heart failure guidelines

Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
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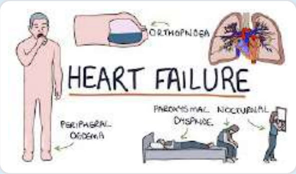
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1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Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

OSMOSIS.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **22.6%**

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An 89-year-old woman presents to the emergency room with increased shortness of breath and decreased mobility of 1-week duration. Her shortness of breath is worse at night and she sometimes wakes up gasping for breath. She has fallen over twice in the last week which isn't normal for her. She has a past medical history of diabetes mellitus type 2 and hypertension. She has had no previous surgeries. She lives independently with her husband and her daughter lives close by and helps with the shopping.

On examination, she is found to have coarse crackles bi-basally on auscultation of her chest. She has a regular heart rate with a pan-systolic murmur loudest over the apex.

Investigations show:

Haemoglobin	11g/dl
WCC	6 x 10^9/l
Platelets	178 x 10^9/l
Sodium	139 mmol/l
Potassium	4.2 mmol/l
Urea	8 mmol/l
Creatinine	92 µmol/l
Blood cultures	*Methicillin-sensitive Staphylococcus aureus*

Echocardiogram severe mitral regurgitation with large mobile structure on valve leaflet

Chest X-ray: bilateral blunting of the costophrenic angles and upper lobe diversion

What is the best treatment for this lady?

○ Flucloxacillin orally

○ Flucloxacillin intravenously ×

○ Amoxicillin orally and vancomycin intravenously ×

○ Ceftriaxone intravenously ×

○ Amoxicillin intravenously and vancomycin intravenously ×

Submit answer

Reference ranges ⌄

Score: **22.6%**

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Echocardiogram severe mitral regurgitation with large mobile structure on valve leaflet

Chest X-ray: bilateral blunting of the costophrenic angles and upper lobe diversion

What is the best treatment for this lady?

Flucloxacillin orally 6%

Flucloxacillin intravenously	70%
Amoxicillin orally and vancomycin intravenously	4%
Ceftriaxone intravenously	2%
Amoxicillin intravenously and vancomycin intravenously	17%

This lady has a native mitral valve endocarditis with a bacteraemia caused by a methicillin-sensitive *Staphylococcus aureus*. Once cultures have grown the causative bacteria and it is found to be sensitive to methicillin guidelines suggest flucloxacillin intravenously to be the treatment of choice. Flucloxacillin orally would not provide sufficient antimicrobial cover nor will ceftriaxone, amoxicillin and vancomycin.

There is some debate about the optimal length of treatment, but 6 weeks of intravenous therapy is generally accepted as the length of treatment needed. Shorter therapeutic regimens may be effective in selected patients with right-sided endocarditis and with endocarditis due to highly susceptible *Streptococcus viridans* treated with synergistic antimicrobials.

https://www.britishinfection.org/files/2714/1640/8773/EndocarditisfinalBSAC2012.pdf

👍 👎 Discuss (3) Improve

Next question >

Infective endocarditis: prognosis and management ★

Poor prognostic factors

- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism

- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	**Suggested antibiotic therapy**
Initial blind therapy	Native valve - amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis - vancomycin + low-dose gentamicin If prosthetic valve - vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA - vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA - vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic - vancomycin + low-dose gentamicin

Scenario	Suggested antibiotic therapy
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic - vancomycin + low-dose gentamicin

Indications for surgery

- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **22.4%**

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Question 198 of 328

An 82-year-old presents with suprapubic discomfort. He reports urgency but has been unable to pass any urine for over 24 hours. Three months ago, he underwent a third failed transurethral resection of the prostate for benign prostatic hypertrophy (PSA was normal). His past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease (baseline creatinine 150 µmol/l) previous MI in 2007 and 2010. He remains relatively active, with an exercise tolerance of 500 yards. On examination, his mucous membranes are moist, his peripheries are warm and his JVP at 3 cm above the angle of Louis. His heart sounds and chest are unremarkable. His abdomen is tender and distended in the suprapubic region. On insertion of a urethral catheter, 900mls of residual urine is noted. His blood tests are as follows:

Hb	115 g/l
Platelets	282 * 10^9/l
WBC	6.8 * 10^9/l

Na^+	144 mmol/l
K^+	5.8 mmol/l
Urea	12.1 mmol/l
Creatinine	201 µmol/l
Troponin T	0.08 (normal <0.03)

His ECG demonstrates left bundle branch block (old) and first-degree heart block at a rate of 49 beats/ minute and regular. What is the appropriate management?

○ Treat as acute coronary syndrome only ×

○ Intravenous fluids only ×

○ Monitor renal function and consider long term catheter only ×

○ Treat as acute coronary syndrome, monitor renal function and consider long term catheter

○ Treat as acute coronary syndrome, intravenous fluids, monitor renal function and consider long term catheter

Submit answer

Reference ranges ⌵

Score: **22.4%**

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Question 198 of 328 ✗

An 82-year-old presents with suprapubic discomfort. He reports urgency but has been unable to pass any urine for over 24 hours. Three months ago, he underwent a third failed transurethral resection of the prostate for benign prostatic hypertrophy (PSA was normal). His past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease (baseline creatinine 150 µmol/l) previous MI in 2007 and 2010. He remains relatively active, with an exercise tolerance of 500 yards. On examination, his mucous membranes are moist, his peripheries are warm and his JVP at 3 cm above the angle of Louis. His heart sounds and chest are unremarkable. His abdomen is tender and distended in the suprapubic region. On insertion of a urethral catheter, 900mls of residual urine is noted. His blood tests are as follows:

Hb	115 g/l
Platelets	282 * 10^9/l
WBC	6.8 * 10^9/l

Na^+	144 mmol/l
K^+	5.8 mmol/l
Urea	12.1 mmol/l
Creatinine	201 µmol/l
Troponin T	0.08 (normal <0.03)

His ECG demonstrates left bundle branch block (old) and first-degree heart block at a rate of 49 beats/ minute and regular. What is the appropriate management?

Treat as acute coronary syndrome only	1%
Intravenous fluids only	8%
Monitor renal function and consider long term catheter only	69%

Treat as acute coronary syndrome, monitor renal function and consider long term catheter 11%

Treat as acute coronary syndrome, intravenous fluids, monitor renal function and consider long term catheter 12%

This patient is a typical elderly admission with multiple co-morbidities involving multiple organ systems. The key to this question is recognising which aspects are important and which are not. The patient is clearly euvolaemic and intravenous fluids at this stage are not required. However, it would be prudent to monitor urine output and if significant amounts are produced, to replace output with oral intake or intravenous input. Troponin is raised but this is in the context of an acute on chronic kidney injury. In the context of no reported chest pain and no new dynamic ECG changes, treatment as an acute coronary syndrome is unnecessary. The patient is at greater risk of post-renal injury and his renal function tests should instead be monitored. A long-term catheter is a reasonable suggestion considering the number of failed TURPs.

👍 👎 Discuss (9) Improve

Next question >

Cardiac enzymes and protein markers ★

Interpretation of the various cardiac enzymes has now largely been superceded by the introduction of troponin T and I. Questions still however commonly appear in exams.

Key points for the exam
- myoglobin is the first to rise
- CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days)

	Begins to rise	**Peak value**	**Returns to normal**
Myoglobin	1-2 hours	6-8 hours	1-2 days

	Begins to rise	Peak value	Returns to normal
CK-MB	2-6 hours	16-20 hours	2-3 days
CK	4-8 hours	16-24 hours	3-4 days
Trop T	4-6 hours	12-24 hours	7-10 days
AST	12-24 hours	36-48 hours	3-4 days
LDH	24-48 hours	72 hours	8-10 days

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

Patient.info 👍 12 👎 4

Cardiac enzymes and protein markers review

UPMC Dept. Pathology 👍 9 👎 1

Diagram of cardiac enzymes

Suggest link

Report broken link

Score: **22.3%**

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A 59 year-old man with history of ischaemic heart disease and type 2 diabetes mellitus is eight hours post right curative hemicolectomy for bowel malignancy. In the surgical high-dependency unit he is noted to be tachycardic on the monitor

On examination the patient appears comfortable. The pulse rate is 200bpm and the blood pressure is 148/79mmHg. Oxygen saturations are 98% on 2L/min nasal oxygen. Capillary refill is 2 seconds.
The chest is clear to auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform at a rate of 200bpm.

Post-operative blood tests reveal:

Hb	131 g/l
Platelets	563 * 10^9/l
WBC	13.4 * 10^9/l
Na^+	141 mmol/l
K^+	4.1 mmol/l
Mg^{++}	0.87 mmol/l
Urea	4.2 mmol/l
Creatinine	121 µmol/l
Bilirubin	23 µmol/l
ALP	109 u/l
ALT	34 u/l
Albumin	33 g/l

What is the most appropriate initial management?

○ Magnesium sulphate 2g IV ×

○ Amiodarone 300mg IV ×

○ Synchronised DC shock

○ Metoprolol 5mg IV

○ Adenosine 6mg IV

Submit answer

Reference ranges

Score: **22.3%**

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Mg^{++}	0.87 mmol/l
Urea	4.2 mmol/l
Creatinine	121 µmol/l
Bilirubin	23 µmol/l
ALP	109 u/l
ALT	34 u/l
Albumin	33 g/l

What is the most appropriate initial management?

Magnesium sulphate 2g IV 21%

Amiodarone 300mg IV 67%

Synchronised DC shock	6%
Metoprolol 5mg IV	2%
Adenosine 6mg IV	5%

This case outlines a description of ventricular tachycardia.

Following the resuscitation council guidelines, in the absence of shock, syncope, myocardial ischaemia or heart failure (which would necessitate DC cardioversion), the most appropriate initial management is 300mg amiodarone intravenously.

Source: https://www.resus.org.uk/resuscitation-guidelines/peri-arrest-arrhythmias/#tachycardia

👍 👎 Discuss (8) Improve

Next question >

Ventricular tachycardia: management ★

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure, syncope) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks.

Drug therapy
- amiodarone: ideally administered through a central line
- lidocaine: use with caution in severe left ventricular impairment
- procainamide

Verapamil should NOT be used in VT.

If drug therapy fails

- electrophysiological study (EPS)
- implant able cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 43 👎 7

2021 Tachycardia algorithm

Suggest link

Report broken link

Score: **22.2%**

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Question 200 of 328 ✓

A 65-year-old obese female with known type 2 diabetes mellitus presents with one-week history of unstable angina. Her troponin was negative and she presented with no dynamic ECG changes. She undergoes an angiogram and a drug-eluting stent was inserted into a 90% stenosis of his LAD. On discharge, what should her antithrombotic regime be?

Aspirin and prasugrel for 1 month, followed by lifelong aspirin	12%
Aspirin and prasugrel for 1 month, followed by lifelong prasugrel	7%
Aspirin and prasugrel for 1 year, followed by lifelong aspirin	79%
Aspirin only, lifelong	2%
Prasugrel only, lifelong	1%

The correct answer for this question is **aspirin and prasugrel for 1 year, followed by lifelong aspirin**. This is because the patient has undergone percutaneous coronary intervention (PCI) with a drug-eluting stent (DES) insertion, which requires dual antiplatelet therapy (DAPT) for at least one year according to UK guidelines. Aspirin is given as a lifelong therapy, while prasugrel, a P2Y12 inhibitor, is given in combination with aspirin for the first year. This regimen helps to prevent stent thrombosis and reduce the risk of recurrent ischemic events.

The option **aspirin and prasugrel for 1 month, followed by lifelong aspirin** is incorrect because DAPT duration of only 1 month is too short after DES placement. The minimum recommended duration of DAPT following DES implantation in patients with stable coronary artery disease or acute coronary syndrome (ACS) without high bleeding risk is 6 months and 12 months respectively.

The option **aspirin and prasugrel for 1 month, followed by lifelong prasugrel** is also incorrect because it does not provide adequate duration of DAPT after DES placement. Moreover, switching from DAPT to monotherapy with only prasugrel would not be appropriate as aspirin remains the cornerstone of long-term antithrombotic therapy in these patients.

Aspirin only, lifelong is not an appropriate option either since it does not include a P2Y12 inhibitor like prasugrel or clopidogrel that should be added to aspirin during the first year following PCI with DES insertion to minimize the risk of stent thrombosis.

Lastly, **prasugrel only, lifelong** would not be suitable as it does not include aspirin, which is a crucial component of long-term antithrombotic therapy in patients with coronary artery disease. Furthermore, prasugrel monotherapy has not been shown to be superior to DAPT with aspirin and a P2Y12 inhibitor in preventing recurrent ischemic events.

In summary, the best antithrombotic regimen for this patient would be **aspirin and prasugrel for 1 year, followed by lifelong aspirin**, as it provides optimal protection against stent thrombosis and recurrent ischemic events following PCI with DES insertion.

👍 👎 💬 Discuss (4) Improve

Next question >

Antiplatelets: summary of latest guidance ★

The table below summarises the most recent guidelines regarding antiplatelets:

Diagnosis	**1st line**	**2nd line**
Acute coronary syndrome (medically treated)	Aspirin (lifelong) & ticagrelor (12 months)	If aspirin contraindicated, clopidogrel (lifelong)
Percutaneous coronary intervention	Aspirin (lifelong) & prasurgrel or ticagrelor (12 months)	If aspirin contraindicated, clopidogrel (lifelong)

Diagnosis	1st line	2nd line
TIA	Clopidogrel (lifelong)	Aspirin (lifelong) & dipyridamole (lifelong)
Ischaemic stroke	Clopidogrel (lifelong)	Aspirin (lifelong) & dipyridamole (lifelong)
Peripheral arterial disease	Clopidogrel (lifelong)	Asprin (lifelong)

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 28 👎 23

2016 Stroke guidelines

Suggest link Report broken link

Media

Thrombolytics

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 1 👎 1

Report broken media

Score: **22.6%**

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Back to top

A 45-year-old man is being worked up for chest pain.

He has a background of bipolar disorder for which he takes olanzapine but is otherwise fit and well.

He has had a cardiac MIBI scan that is reported as follows:

Images display a moderate perfusion abnormality in the right ventricle on the stress images with associated dyskinesis. This defect is also present at rest.

Given the above information, what is the most likely cause of these imaging findings?

- ○ Atherosclerosis
- ○ Hypertrophic cardiomyopathy
- ○ Kawasaki disease
- ○ Soft tissue attenuation defect
- ○ Transmural scarring

Submit answer

Reference ranges

Score: **0%**

1 -

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Question 1 of 129

A 45-year-old man is being worked up for chest pain.

He has a background of bipolar disorder for which he takes olanzapine but is otherwise fit and well.

He has had a cardiac MIBI scan that is reported as follows:

Images display a moderate perfusion abnormality in the right ventricle on the stress images with associated dyskinesis. This defect is also present at rest.

Given the above information, what is the most likely cause of these imaging findings?

Atherosclerosis	28%
Hypertrophic cardiomyopathy	11%
Kawasaki disease	3%
Soft tissue attenuation defect	14%
Transmural scarring	45%

If a cardiac MIBI scan shows that the defect is present on both stress and rest, this is suggestive of a fixed defect such as myocardial necrosis and fibrosis secondary to infarction

Important for me Less important

Here we have a patient with a perfusion defect in the right ventricle at rest and during stress. This indicated that the lesion was not reversible.

Transmural scarring is correct. When scar tissue is formed within the myocardium, there will be a fixed perfusion defect as blood supply to this area never normalises.

Atherosclerosis is incorrect. Atherosclerosis commonly causes reversible defects that normalise at rest.

Soft tissue attenuation defect is incorrect. Although soft tissue attenuation defects can cause the appearance of fixed defects, there would not be associated wall motion abnormalities.

Kawasaki disease is incorrect. Although this vasculitis can be a cause of myocardial infarction it is very rare. It usually affects children under the age of five and is associated with fever, strawberry tongue, rash, and lymphadenopathy, none of which are seen in this case.

Hypertrophic cardiomyopathy is incorrect. This would be visible on MIBI scan as ventricular hypertrophy, if a defect is present, it is likely to be present only during stress.

👍 👎 Discuss (3) Improve

Next question >

Cardiac imaging: non-invasive techniques excluding echocardiography ★

The ability to image the heart using non-invasive techniques such as MRI, CT and radionuclides has evolved rapidly over recent years.

Nuclear imaging

These techniques use radiotracers which are extracted by normal myocardium. Examples include:
- thallium
- technetium (99mTc) sestamibi: a coordination complex of the radioisotope technetium-99m with the ligand methoxyisobutyl isonitrile (MIBI), used in 'MIBI' or cardiac Single Photon Emission Computed Tomography (SPECT) scans
- fluorodeoxyglucose (FDG): used in Positron Emission Tomography (PET) scans

The primary role of SPECT is to assess myocardial perfusion and myocardial viability. Two sets of images are usually acquired. First the myocardium at rest followed by images of the myocardium during stress (either exercise or following adenosine / dipyridamole). By comparing the rest with stress images any areas of ischaemia can classified as reversible or fixed (e.g. Following a myocardial infarction). Cardiac PET is predominately a research tool at the current time

MUGA

- Multi Gated Acquisition Scan, also known as radionuclide angiography
- radionuclide (technetium-99m) is injected intravenously
- the patient is placed under a gamma camera
- may be performed as a stress test
- can accurately measure left ventricular ejection fraction. Typically used before and after cardiotoxic drugs are used

Cardiac Computed Tomography (CT)

Cardiac CT is useful for assessing suspected ischaemic heart disease, using two main methods:

- calcium score: there is known to be a correlation between the amount of atherosclerotic plaque calcium and the risk of future ischaemic events. Cardiac CT can quantify the amount of calcium producing a 'calcium score'
- contrast enhanced CT: allows visualisation of the coronary artery lumen

If these two techniques are combined cardiac CT has a very high negative predictive value for ischaemic heart disease.

Cardiac MRI

Cardiac MRI (commonly termed CMR) has become the gold standard for providing structural images of the heart. It is particularly useful when assessing congenital heart disease, determining right and left ventricular mass and differentiating forms of cardiomyopathy. Myocardial perfusion can also be assessed following the administration of gadolinium. Currently CMR provides limited data on the extent of coronary artery disease.

Please also see the British Heart Foundation link for an excellent summary.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 2 👎 4

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Royal College of Physicians 👍 3 👎 2

2012 Nuclear cardiology review

Suggest link Report broken link

Score: **0%**

1 ✗

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Question 2 of 129

A 69-year-old man presents with central crushing chest pain. He also feels nauseous. He states that the pain came on at rest. He has a past medical history of hypertension and poorly controlled type 2 diabetes mellitus.

An ECG is performed:

P waves	Normal morphology
PR interval	120ms
QRS	115ms
T waves	Deeply inverted T waves in V2-V3
ST segments	No elevation or depression

What is the most likely diagnosis?

- ○ Critical stenosis of the right coronary artery ×
- ○ Critical stenosis of the left main stem coronary artery ×
- ○ Critical stenosis of the left anterior descending artery (LAD) ×
- ○ 75% stenosis of the left anterior descending artery (LAD) ×
- ○ 75% stenosis of the right coronary artery ×

Submit answer

Reference ranges

Score: **0%**

1	✗
2	-

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A 69-year-old man presents with central crushing chest pain. He also feels nauseous. He states that the pain came on at rest. He has a past medical history of hypertension and poorly controlled type 2 diabetes mellitus.

An ECG is performed:

P waves	Normal morphology
PR interval	120ms
QRS	115ms
T waves	Deeply inverted T waves in V2-V3
ST segments	No elevation or depression

What is the most likely diagnosis?

Critical stenosis of the right coronary artery	4%
Critical stenosis of the left main stem coronary artery	7%
Critical stenosis of the left anterior descending artery (LAD)	59%
75% stenosis of the left anterior descending artery (LAD)	27%
75% stenosis of the right coronary artery	3%

Wellens' syndrome is a pattern of deeply inverted or biphasic T waves in V2-3. It is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation

Important for me Less important

The patient has multiple major risk factors for cardiovascular disease making an acute coronary syndrome high up the differential list. The deeply inverted T waves in V2-V3 are very suggestive of Wellens' syndrome which is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation.

Right coronary artery pathology tends to present with ECG changes in the inferior leads (II, III and AVF).

Left main stem coronary artery occlusion tends to present with widespread ECG changes with horizontal ST depression (most prominent in leads I, II and V4-6), and ST elevation in aVR.

👍 | 👎 | Discuss (9) | Improve

Next question ›

Acute coronary syndrome: initial management

★

Acute coronary syndrome (ACS) is a very common and important presentation in medicine. The management of ACS has evolved over recent years, with the development of new drugs and procedures such as percutaneous coronary intervention (PCI).

Emergency departments often have their own protocols based on local factors such as the availability of PCI and hospital drug formularies. The following is based on the 2020 update to the NICE ACS guidelines.

Acute coronary syndrome can be classified as follows:
- ST-elevation myocardial infarction (STEMI): ST-segment elevation + elevated biomarkers of myocardial damage
- non ST-elevation myocardial infarction (NSTEMI): ECG changes but no ST-segment elevation + elevated biomarkers of myocardial damage
- unstable angina

The management of ACS depends on the particular subtype. NICE management guidance groups the patients into two groups:

- 1. STEMI
- 2. NSTEM/unstable angina

Common management of all patients with ACS

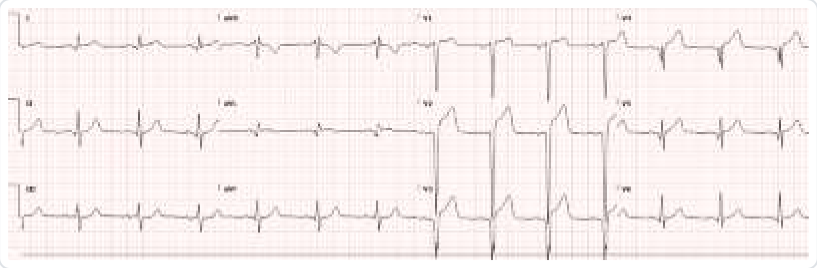
Initial drug therapy

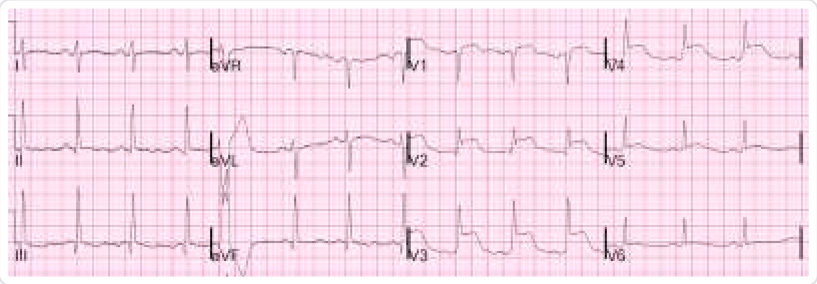
- aspirin 300mg
- oxygen should only be given if the patient has oxygen saturations < 94% in keeping with British Thoracic Society oxygen therapy guidelines
- morphine should only be given for patients with severe pain
  - previously IV morphine was given routinely
  - evidence, however, suggests that this may be associated with adverse outcomes
- nitrates
  - can be given either sublingually or intravenously
  - useful if the patient has ongoing chest pain or hypertension
  - should be used in caution if patient hypotensive

The next step in managing a patient with suspected ACS is to determine whether they meet the ECG criteria for STEMI. It is, of course, important to recognise that these criteria should be interpreted in the context of the clinical history.

STEMI criteria

- clinical symptoms consistent with ACS (generally of ≥ 20 minutes duration) with persistent (> 20 minutes) ECG features in ≥ 2 contiguous leads of:
  - 2.5 mm (i.e ≥ 2.5 small squares) ST elevation in leads V2-3 in men under 40 years, or ≥ 2.0 mm (i.e ≥ 2 small squares) ST elevation in leads V2-3 in men over 40 years
  - 1.5 mm ST elevation in V2-3 in women
  - 1 mm ST elevation in other leads
  - new LBBB (LBBB should be considered new unless there is evidence otherwise)

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Management of STEMI

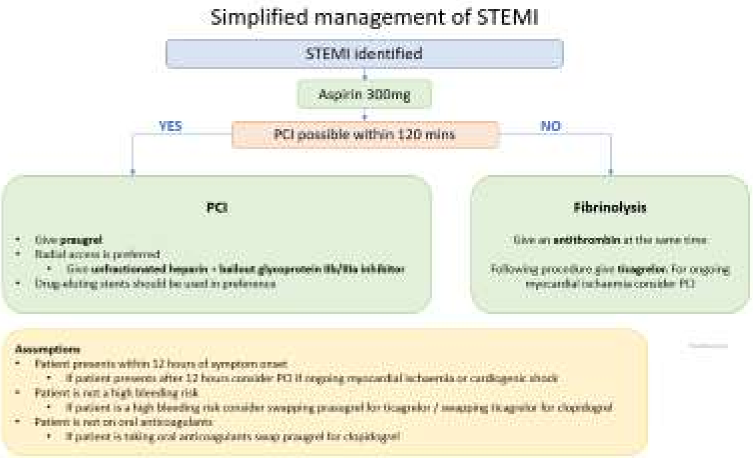
Simplified management of STEMI
STEMI identified
Aspirin 300mg
YES
PCI possible within 120 mins
NO
PCI
Give prasugrel
Radial access is preferred
Give unfractionated heparin + bailout glycoprotein IIb/IIIa inhibitor
Drug-eluting stents should be used in preference
Fibrinolysis
Give an antithrombin at the same time
Following procedure give ticagrelor. For ongoing myocardial ischaemia consider PCI
Assumptions
Patient presents within 12 hours of symptom onset
If patient presents after 12 hours consider PCI if ongoing myocardial ischaemia or cardiogenic shock
Patient is not a high bleeding risk
If patient is a high bleeding risk consider swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel for clopidogrel

Diagram showing the simplified management of STEMI according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

Once a STEMI has been confirmed the first step is to immediately assess eligibility for coronary reperfusion therapy. There are two types of coronary reperfusion therapy:

- percutaneous coronary intervention
  - should be offered if the presentation is within 12 hours of the onset of symptoms AND PCI can be delivered within 120 minutes of the time when fibrinolysis could have been given (i.e. consider fibrinolysis if there is a significant delay in being able to provide PCI)
  - if patients present after 12 hours and still have evidence of ongoing ischaemia then PCI should still be considered

    - drug-eluting stents are now used. Previously 'bare-metal' stents were sometimes used but have higher rates of restenosis
    - radial access is preferred to femoral access
- fibrinolysis
    - should be offered within 12 hours of the onset of symptoms if primary PCI cannot be delivered within 120 minutes of the time when fibrinolysis could have been given
    - a practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) that does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI

If patients are eligible this should be offered as soon as possible.

Percutaneous coronary intervention for patients with STEMI

Further antiplatelet prior to PCI

- this is termed 'dual antiplatelet therapy', i.e. aspirin + another drug
- if the patient is not taking an oral anticoagulant: prasugrel
- if taking an oral anticoagulant: clopidogrel

Drug therapy during PCI

- patients undergoing PCI with radial access:
    - unfractionated heparin with bailout glycoprotein IIb/IIIa inhibitor (GPI) - this is the action of using a GPI during the procedure when it was not intended from the outset, e.g. because of worsening or persistent thrombus
- patients undergoing PCI with femoral access:
    - bivalirudin with bailout GPI

Other procedures during PCI

- thrombus aspiration, but not mechanical thrombus extraction, should be considered
- complete revascularisation should be considered for patients with multivessel coronary artery disease without cardiogenic shock

Fibrinolysis for patients with STEMI

Fibrinolysis used to be the only form of coronary reperfusion therapy available. However, it is used much less commonly now given the widespread availability of PCI.

The contraindications to fibrinolysis and other factors are described in other notes.

Patients undergoing fibrinolysis should also be given an antithrombin drug.

An ECG should be repeated after 60-90 minutes to see if the ECG changes have resolved. If patients have persistent myocardial ischaemia following fibrinolysis then PCI should be considered.

Management of NSTEMI/unstable angina

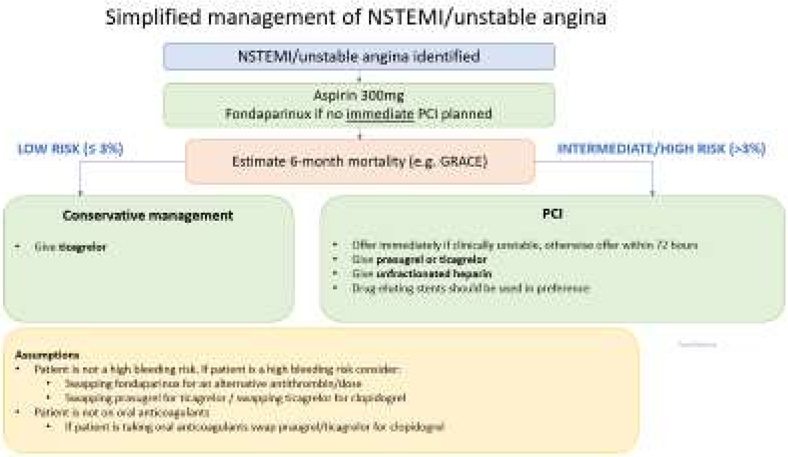
Simplified management of NSTEMI/unstable angina
NSTEMI/unstable angina identified
Aspirin 300mg
Fondaparinux if no immediate PCI planned
LOW RISK (≤ 3%)
Estimate 6-month mortality (e.g. GRACE)
INTERMEDIATE/HIGH RISK (>3%)
Conservative management
Give ticagrelor
PCI
Offer immediately if clinically unstable, otherwise offer within 72 hours
Give prasugrel or ticagrelor
Give unfractionated heparin
Drug-eluting stents should be used in preference
Assumptions
Patient is not a high bleeding risk. If patient is a high bleeding risk consider:
Swapping fondaparinux for an alternative antithrombin/dose
Swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel/ticagrelor for clopidogrel

Diagram showing the simplified management of NSTEMI/unstable angina according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

The management of NSTEMI/unstable angina is complicated and depends on individual patient factors and a risk assessment. The summary below provides an overview but the full NICE guidelines should be reviewed for further details.

Further drug therapy

- antithrombin treatment
  - fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography immediately

    - if immediate angiography is planned or a patients creatinine is > 265 µmol/L then unfractionated heparin should be given

Risk assessment

The Global Registry of Acute Coronary Events (GRACE) is the most widely used tool for risk assessment. It can be calculated using online tools and takes into account the following factors:
- age
- heart rate, blood pressure
- cardiac (Killip class) and renal function (serum creatinine)
- cardiac arrest on presentation
- ECG findings
- troponin levels

This results in the patient being risk stratified as follows:

Predicted 6-month mortality	Risk of future adverse cardiovascular events
1.5% or below	Lowest
> 1.5% to 3.0%	Low
> 3.0% to 6.0%	Intermediate
> 6.0% to 9.0%	High
over 9.0%	Highest

Based on this risk assessment key decisions are made regarding whether a patient has coronary angiography (with follow-on PCI if necessary) or has conservative management. The detailed pros/cons of this decision are covered in other notes.

Which patients with NSTEMI/unstable angina should have coronary angiography (with follow-on PCI if necessary)?
- immediate: patient who are clinically unstable (e.g. hypotensive)
- within 72 hours: patients with a GRACE score > 3% i.e. those at intermediate, high or highest risk
- coronary angiography should also be considered for patients if ischaemia is subsequently experienced after admission

Percutaneous coronary intervention for patients with NSTEMI/unstable angina

Further drug therapy
- unfractionated heparin should be given regardless of whether the patient has had fondaparinux or not
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug) prior to PCI
  - if the patient is not taking an oral anticoagulant: prasugrel or ticagrelor
  - if taking an oral anticoagulant: clopidogrel

Conservative management for patients with NSTEMI/unstable angina

Further drug therapy
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug)
  - if the patient is not at a high risk of bleeding: ticagrelor
  - if the patient is at a high risk of bleeding: clopidogrel

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 👍 24 👎 28

2016 Acute coronary syndrome guidelines

NICE 👍 64 👎 34

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Media

Acute Coronary Syndrome DETAILED Overview (MI, STEMI, NSTEMI)

Armando Hasudungan - YouTube 👍 5 👎 0

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 4 👎 1

Report broken media

Score: **50%**

1 ✗

2 ✓

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Question 3 of 129

A 28-year-old man is admitted with 4 episodes of shocks from his ICD in the past 24 hours. He states that the chest pain is like an electric shock, which is brief and lasts for only a second or two. He also complains of palpitations. He has a past medical history of hypertrophic obstructive cardiomyopathy (HOCM) and has an ICD due to previous episodes of ventricular tachycardia.

Whilst in the emergency department he suffers from 8 consecutive shocks from the ICD. A 12 lead ECG shows an irregularly irregular rhythm with a QRS of 0.10s, and absence of P waves. His heart rate is 140bpm and blood pressure = 130/75 mmHg. There is no signs of heart failure on examination.

What is the most appropriate initial management?

- ○ Apply a ring magnet over the ICD to temporarily deactivate it pending device interrogation
- ○ Rate control with calcium channel blocker
- ○ Chemical cardioversion with amiodarone
- ○ Synchronised electrical cardioversion
- ○ Rate control with beta blocker

Submit answer

Reference ranges

Score: **50%**

1	✗
2	✓
3	-

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A 28-year-old man is admitted with 4 episodes of shocks from his ICD in the past 24 hours. He states that the chest pain is like an electric shock, which is brief and lasts for only a second or two. He also complains of palpitations. He has a past medical history of hypertrophic obstructive cardiomyopathy (HOCM) and has an ICD due to previous episodes of ventricular tachycardia.

Whilst in the emergency department he suffers from 8 consecutive shocks from the ICD. A 12 lead ECG shows an irregularly irregular rhythm with a QRS of 0.10s, and absence of P waves. His heart rate is 140bpm and blood pressure = 130/75 mmHg. There is no signs of heart failure on examination.

What is the most appropriate initial management?

Apply a ring magnet over the ICD to temporarily deactivate it pending device interrogation	46%
Rate control with calcium channel blocker	3%
Chemical cardioversion with amiodarone	20%
Synchronised electrical cardioversion	7%
Rate control with beta blocker	24%

Multiple episodes of inappropriate shocks from an ICD can be both unpleasant and dangerous. Ultimately the device will need interrogated from a pacemaker technician, however the most immediate management should be to place a ring magnet over the ICD to prevent further inappropriate shocks.

Important for me Less important

The patient has presented with multiple episodes of shocks from his ICD. The ICD was inserted to cardiovert life threatening arrhythmias such as ventricular tachycardia and fibrillation. However in this case, he has developed new onset AF, and has received multiple inappropriate shocks from his ICD.

Ultimately he will need the device interrogated from a pacemaker technician, however the most immediate management should be to place a ring magnet over the ICD to prevent further inappropriate shock, which can both be painful, and cause harm. This must only be done while the patient is on a cardiac monitor or telemetry so that one can monitor for any life threatening arrhythmias. If, at any time, you identify ventricular tachycardia or ventricular fibrillation on the cardiac monitor, the ring magnet must be removed immediately allowing normal firing of the ICD to resume after approximately 15 seconds.

One could argue that pharmacological cardioversion or rate control would resolve the problem. However the most immediate management would involve preventing further shocks with a ring magnet while the device is interrogated.

👍 👎 Discuss (10) Improve

Next question >

Implantable cardiac defibrillators ★

Indications
- long QT syndrome
- hypertrophic obstructive cardiomyopathy
- previous cardiac arrest due to VT/VF
- previous myocardial infarction with non-sustained VT on 24 hr monitoring, inducible VT on electrophysiology testing and ejection fraction < 35%
- Brugada syndrome

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **33.3%**

1	✗
2	✓
3	✗

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Question 4 of 129

A 48-year-old hypertensive, male who smokes presents with central crushing chest pain and nausea. His ECG shows new deeply inverted T waves in V2 and V3.

These ECG findings are consistent with critical stenosis to which coronary artery?

- ○ Left anterior descending artery ×
- ○ Left circumflex artery ×
- ○ Left main artery ×
- ○ Posterior descending artery ×
- ○ Right coronary artery ×

Submit answer

Reference ranges

Score: **33.3%**

1	✗
2	✓
3	✗
4	-

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A 48-year-old hypertensive, male who smokes presents with central crushing chest pain and nausea. His ECG shows new deeply inverted T waves in V2 and V3.

These ECG findings are consistent with critical stenosis to which coronary artery?

Left anterior descending artery	78%
Left circumflex artery	6%
Left main artery	5%
Posterior descending artery	8%
Right coronary artery	3%

Wellens' syndrome is a pattern of deeply inverted or biphasic T waves in V2-3. It is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation

Important for me Less important

This patient presents with typical symptoms of acute coronary syndrome. Deeply inverted or biphasic T waves in V2 and V3 are consistent with a diagnosis of Wellens' syndrome. Wellens' syndrome is caused by critical stenosis of the left anterior descending (LAD) artery and is associated with a high risk of extensive anterior wall myocardial infarction. Patients may be pain-free at presentation or have minimally elevated cardiac enzymes. Patients with Wellens' syndrome typically require intervention with urgent angiogram and revascularisation, as in an acute ST-elevation myocardial infarction. The other listed coronary arteries are not associated with Wellens' syndrome.

The left circumflex artery supplies the left ventricle.

The left main artery supplies the left ventricle and atrium.

The posterior descending artery supplies the inferior aspect of the myocardium.

The right coronary artery supplies the right ventricle, right atrium and the sino-atrial and atrioventricular nodes of the heart.

👍 | 👎 | Discuss (3) | Improve

Next question ›

Acute coronary syndrome: initial management

★

Acute coronary syndrome (ACS) is a very common and important presentation in medicine. The management of ACS has evolved over recent years, with the development of new drugs and procedures such as percutaneous coronary intervention (PCI).

Emergency departments often have their own protocols based on local factors such as the availability of PCI and hospital drug formularies. The following is based on the 2020 update to the NICE ACS guidelines.

Acute coronary syndrome can be classified as follows:

- ST-elevation myocardial infarction (STEMI): ST-segment elevation + elevated biomarkers of myocardial damage
- non ST-elevation myocardial infarction (NSTEMI): ECG changes but no ST-segment elevation + elevated biomarkers of myocardial damage
- unstable angina

The management of ACS depends on the particular subtype. NICE management guidance groups the patients into two groups:

- 1. STEMI
- 2. NSTEM/unstable angina

Common management of all patients with ACS

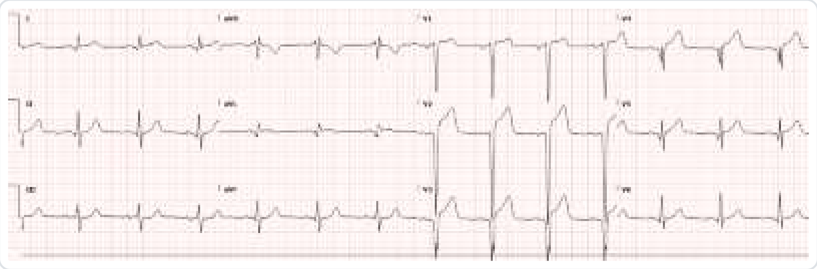
Initial drug therapy

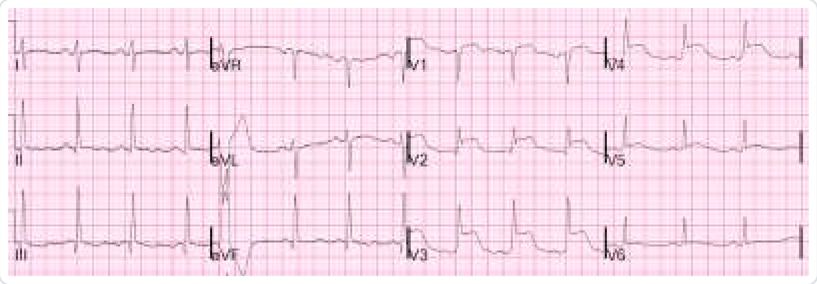
- aspirin 300mg
- oxygen should only be given if the patient has oxygen saturations < 94% in keeping with British Thoracic Society oxygen therapy guidelines
- morphine should only be given for patients with severe pain
    - previously IV morphine was given routinely
    - evidence, however, suggests that this may be associated with adverse outcomes
- nitrates
    - can be given either sublingually or intravenously
    - useful if the patient has ongoing chest pain or hypertension
    - should be used in caution if patient hypotensive

The next step in managing a patient with suspected ACS is to determine whether they meet the ECG criteria for STEMI. It is, of course, important to recognise that these criteria should be interpreted in the context of the clinical history.

STEMI criteria

- clinical symptoms consistent with ACS (generally of ≥ 20 minutes duration) with persistent (> 20 minutes) ECG features in ≥ 2 contiguous leads of:
    - 2.5 mm (i.e ≥ 2.5 small squares) ST elevation in leads V2-3 in men under 40 years, or ≥ 2.0 mm (i.e ≥ 2 small squares) ST elevation in leads V2-3 in men over 40 years
    - 1.5 mm ST elevation in V2-3 in women
    - 1 mm ST elevation in other leads
    - new LBBB (LBBB should be considered new unless there is evidence otherwise)




I
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Management of STEMI

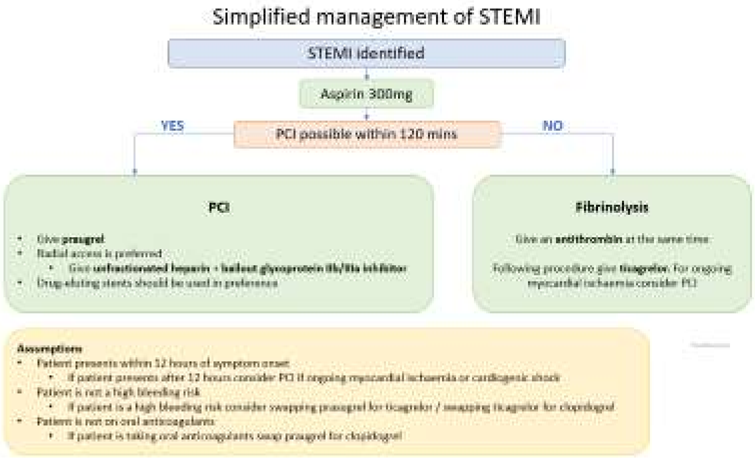
Simplified management of STEMI
STEMI identified
Aspirin 300mg
PCI possible within 120 mins
YES
NO
PCI
Give prasugrel
Radial access is preferred
Give unfractionated heparin + bailout glycoprotein IIb/IIIa inhibitor
Drug-eluting stents should be used in preference
Fibrinolysis
Give an antithrombin at the same time
Following procedure give ticagrelor. For ongoing myocardial ischaemia consider PCI
Assumptions
Patient presents within 12 hours of symptom onset
If patient presents after 12 hours consider PCI if ongoing myocardial ischaemia or cardiogenic shock
Patient is not a high bleeding risk
If patient is a high bleeding risk consider swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel for clopidogrel

Diagram showing the simplified management of STEMI according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

Once a STEMI has been confirmed the first step is to immediately assess eligibility for coronary reperfusion therapy. There are two types of coronary reperfusion therapy:

- percutaneous coronary intervention
    - should be offered if the presentation is within 12 hours of the onset of symptoms AND PCI can be delivered within 120 minutes of the time when fibrinolysis could have been given (i.e. consider fibrinolysis if there is a significant delay in being able to provide PCI)
    - if patients present after 12 hours and still have evidence of ongoing ischaemia then PCI should still be considered
    - drug-eluting stents are now used. Previously 'bare-metal' stents were sometimes used but have higher rates of restenosis
    - radial access is preferred to femoral access
- fibrinolysis
    - should be offered within 12 hours of the onset of symptoms if primary PCI cannot be delivered within 120 minutes of the time when fibrinolysis could have been given

  - a practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) that does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI

If patients are eligible this should be offered as soon as possible.

Percutaneous coronary intervention for patients with STEMI

Further antiplatelet prior to PCI
- this is termed 'dual antiplatelet therapy', i.e. aspirin + another drug
- if the patient is not taking an oral anticoagulant: prasugrel
- if taking an oral anticoagulant: clopidogrel

Drug therapy during PCI
- patients undergoing PCI with radial access:
  - unfractionated heparin with bailout glycoprotein IIb/IIIa inhibitor (GPI) - this is the action of using a GPI during the procedure when it was not intended from the outset, e.g. because of worsening or persistent thrombus
- patients undergoing PCI with femoral access:
  - bivalirudin with bailout GPI

Other procedures during PCI
- thrombus aspiration, but not mechanical thrombus extraction, should be considered
- complete revascularisation should be considered for patients with multivessel coronary artery disease without cardiogenic shock

Fibrinolysis for patients with STEMI

Fibrinolysis used to be the only form of coronary reperfusion therapy available. However, it is used much less commonly now given the widespread availability of PCI.

The contraindications to fibrinolysis and other factors are described in other notes.

Patients undergoing fibrinolysis should also be given an antithrombin drug.

An ECG should be repeated after 60-90 minutes to see if the ECG changes have resolved. If patients have persistent myocardial ischaemia following fibrinolysis then PCI should be considered.

Management of NSTEMI/unstable angina

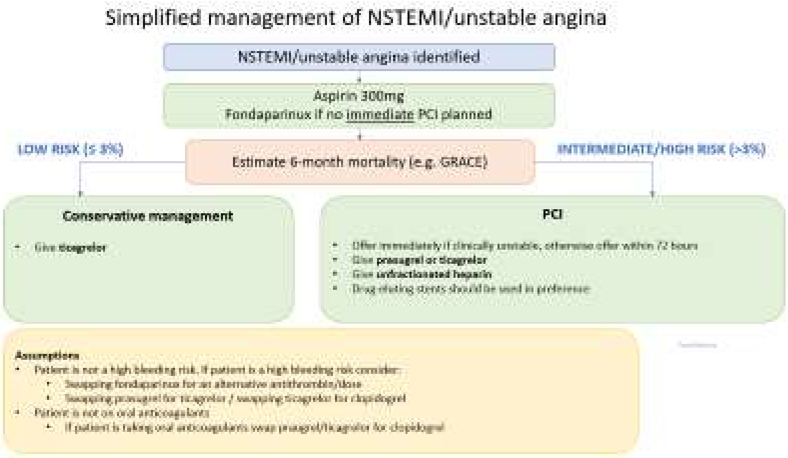
Simplified management of NSTEMI/unstable angina
NSTEMI/unstable angina identified
Aspirin 300mg
Fondaparinux if no immediate PCI planned
LOW RISK (≤ 3%)
Estimate 6-month mortality (e.g. GRACE)
INTERMEDIATE/HIGH RISK (>3%)
Conservative management
Give ticagrelor
PCI
Offer immediately if clinically unstable, otherwise offer within 72 hours
Give prasugrel or ticagrelor
Give unfractionated heparin
Drug eluting stents should be used in preference
Assumptions
Patient is not a high bleeding risk. If patient is a high bleeding risk consider:
Swapping fondaparinux for an alternative antithrombin/dose
Swapping prasugrel for ticagrelor / swapping ticagrelor for clopidogrel
Patient is not on oral anticoagulants
If patient is taking oral anticoagulants swap prasugrel/ticagrelor for clopidogrel

Diagram showing the simplified management of NSTEMI/unstable angina according to NICE guidelines. A number of assumptions (listed at the bottom) are made.

The management of NSTEMI/unstable angina is complicated and depends on individual patient factors and a risk assessment. The summary below provides an overview but the full NICE guidelines should be reviewed for further details.

Further drug therapy

- antithrombin treatment
  - fondaparinux should be offered to patients who are not at a high risk of bleeding and who are not having angiography immediately
  - if immediate angiography is planned or a patients creatinine is > 265 µmol/L then unfractionated heparin should be given

Risk assessment

The Global Registry of Acute Coronary Events (GRACE) is the most widely used tool for risk assessment. It can be calculated using online tools and takes into account the following factors:

- age
- heart rate, blood pressure
- cardiac (Killip class) and renal function (serum creatinine)
- cardiac arrest on presentation
- ECG findings
- troponin levels

This results in the patient being risk stratified as follows:

Predicted 6-month mortality	Risk of future adverse cardiovascular events
1.5% or below	Lowest
> 1.5% to 3.0%	Low
> 3.0% to 6.0%	Intermediate
> 6.0% to 9.0%	High
over 9.0%	Highest

Based on this risk assessment key decisions are made regarding whether a patient has coronary angiography (with follow-on PCI if necessary) or has conservative management. The detailed pros/cons of this decision are covered in other notes.

Which patients with NSTEMI/unstable angina should have coronary angiography (with follow-on PCI if necessary)?

- immediate: patient who are clinically unstable (e.g. hypotensive)
- within 72 hours: patients with a GRACE score > 3% i.e. those at intermediate, high or highest risk
- coronary angiography should also be considered for patients if ischaemia is subsequently experienced after admission

Percutaneous coronary intervention for patients with NSTEMI/unstable angina

Further drug therapy

- unfractionated heparin should be given regardless of whether the patient has had fondaparinux or not
- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug) prior to PCI
    - if the patient is not taking an oral anticoagulant: prasugrel or ticagrelor
    - if taking an oral anticoagulant: clopidogrel

Conservative management for patients with NSTEMI/unstable angina

Further drug therapy

- further antiplatelet ('dual antiplatelet therapy', i.e. aspirin + another drug)
    - if the patient is not at a high risk of bleeding: ticagrelor
    - if the patient is at a high risk of bleeding: clopidogrel

+ Q 123

Next question >

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Links

SIGN 👍 24 👎 28

2016 Acute coronary syndrome guidelines

NICE 👍 64 👎 34

2020 Acute coronary syndromes guidelines

Suggest link Report broken link

Media

Acute Coronary Syndrome DETAILED Overview (MI, STEMI, NSTEMI)

Armando Hasudungan - YouTube 👍 5 👎 0

Antiplatelets and anticoagulants made easy

Speed Pharmacology - YouTube 👍 4 👎 1

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Score: **25%**

1	✗
2	✓
3	✗
4	✗

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Question 5 of 129

An 8-year-old boy presents to the emergency department with seven days of fever. He originally attended six days previously and was discharged home with advice to return if the fever persisted. There has been no response to paracetamol treatment. He has not past medical history.

Observations:

- Heart rate 84 beats per minute
- Blood pressure 100/70 mmHg
- Temperature 39.4C

On examination, his conjunctivae are erythematous. He has red, cracked lips and a 'strawberry tongue'. There is unilateral cervical lymphadenopathy. The palms of his hands and soles of his feet are red. The examination is otherwise normal.

Urinalysis and a chest x-ray are unremarkable.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.9 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	35 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)

Albumin	36 g/L	(35 - 50)

In addition to aspirin, what other treatment has the most evidence for benefit in treating this condition?

- ○ Antibiotic treatment ×
- ○ Cyclophosphamide ×
- ○ Intravenous immunoglobulin ×
- ○ Prednisolone ×
- ○ Rituximab ×

Submit answer

Reference ranges

Score: **25%**

1 ✗
2 ✓
3 ✗
4 ✗
5 -

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Hb	136 g/L	Male: (135-180) Female: (115 - 160)
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Na^+	140 mmol/L	(135 - 145)
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ALP	88 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)

Albumin	36 g/L	(35 - 50)

In addition to aspirin, what other treatment has the most evidence for benefit in treating this condition?

Antibiotic treatment	11%
Cyclophosphamide	2%
Intravenous immunoglobulin	51%
Prednisolone	34%
Rituximab	2%

Intravenous immunoglobulin , uses include: Kawasaki disease

Important for me Less important

Intravenous immunoglobulin is correct. The child has Kawasaki disease as evidenced by a fever lasting > 5 days, bilateral conjunctival congestion, cervical lymphadenopathy and changes of the lips and extremities. This is a medium-vessel vasculitis that occurs in children. In addition to aspirin, intravenous immunoglobulin should be administered to prevent the formation of coronary artery aneurysms, which is a feared complication of this condition.

Antibiotic treatment is incorrect. The clinical syndrome, in this case, suggests Kawasaki disease as the cause of fever, rather than infection. This is an inflammatory rather than infective condition and does not require antibiotic treatment.

Cyclophosphamide is incorrect. This has been used occasionally in refractory Kawasaki disease but it has less evidence than IVIG. Additionally, there is a risk of infertility associated with this treatment and therefore it should be avoided in people of child-bearing age unless absolutely necessary.

Prednisolone is incorrect. There is more limited evidence for the benefit of corticosteroids in this condition compared with IVIG. Adjunctive treatment with corticosteroids could be considered in particular high risk groups (Age < 12 months, Asian ethnicity) and in the setting if particular blood test abnormalities (ALT >100 IU/L, albumin ≤30 g/L, sodium ≤133 mmol/L, platelets ≤30 x 109/L, CRP >100 mg/L, anemia) or cardiac or coronary artery involvement on echo at presentation.

Rituximab is incorrect. This is an anti-CD20 monoclonal antibody that is used in the treatment of some vasculitis. There is limited evidence for its use in Kawasaki disease but it has been used occasionally in IVIG refractory disease.

👍 | 👎 | Discuss (3) | Improve

Next question >

Kawasaki disease

Kawasaki disease is a type of vasculitis which is predominately seen in children. Whilst Kawasaki disease is uncommon it is important to recognise as it may cause potentially serious complications, including coronary artery aneurysms.

Features
- high-grade fever which lasts for > 5 days. Fever is characteristically resistant to antipyretics
- conjunctival injection
- bright red, cracked lips
- strawberry tongue
- cervical lymphadenopathy
- red palms of the hands and the soles of the feet which later peel

Kawasaki disease is a clinical diagnosis as there is no specific diagnostic test.

Management
- high-dose aspirin
  - Kawasaki disease is one of the few indications for the use of aspirin in children. Due to the risk of Reye's syndrome aspirin is normally contraindicated in children
- intravenous immunoglobulin

- echocardiogram (rather than angiography) is used as the initial screening test for coronary artery aneurysms

Complications
- coronary artery aneurysm

123

Next question >

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Media

Kawasaki Disease
Khan Academy

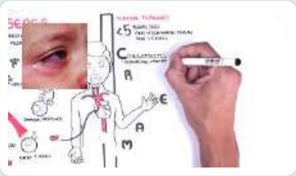
Kawasaki disease

Khan Academy - YouTube 👍 2 👎 0

KAWASAKI DISEASE
~ CLINICAL REASONING ~
Osmosis.org

Kawasaki disease

Osmosis - YouTube 👍 2 👎 0



Kawasaki disease

Armando Hasudungan - YouTube 👍 1 👎 1

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Score: **40%**

1 ✗
2 ✓
3 ✗
4 ✗
5 ✓

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Question 6 of 129

A 67-year-old woman attends Cardiology clinic. She has recently had a permanent pacemaker (PPM) inserted due to pre-syncopal episodes associated with bradycardia. She reports an improvement in these symptoms since insertion, but notes exertional chest pain since the procedure. She has a past medical history of type 2 diabetes mellitus and hypercholesterolaemia, for which she takes metformin and atorvastatin.

What is the most cause of her symptoms?

- ○ Pacemaker lead malposition ×
- ○ Costochondritis ×
- ○ Bornholm disease ×
- ○ Pacemaker syndrome ×
- ○ Coronary ischaemia ×

Submit answer

Reference ranges

Score: **40%**

1	✗
2	✓
3	✗
4	✗
5	✓

6	-

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Question 6 of 129 ✓

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What is the most cause of her symptoms?

Pacemaker lead malposition	9%
Costochondritis	8%
Bornholm disease	11%
Pacemaker syndrome	51%
Coronary ischaemia ✓	21%

Pacemaker lead malposition, costochondritis and Bornholm disease (a viral infection causing pain in the lower chest) would not typically be associated with specifically exertional chest pain.

Pacemaker syndrome is typically associated with a VVI pacemaker that results in simultaneous atria and ventricle conduction. This can cause fatigue, dizziness and hypotension.

This patient is a good candidate for coronary artery disease with type 2 diabetes mellitus and hypercholesterolaemia. Pre-pacemaker insertion her heart had lower energy requirements due to conduction system disease. Pacing has improved her potential heart rate and therefore increased energy demands. This has unmasked her underlying coronary ischaemia. She now needs an angiogram!

👍 👎 Discuss (6) Improve

Next question ›

Chest pain: assessment of patients with suspected cardiac chest pain ★

NICE updated it's guidelines in 2016 on the 'Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin'.

Below is a brief summary of the key points. Please see the link for more details.

Patients presenting with acute chest pain

Immediate management of suspected acute coronary syndrome (ACS)

- glyceryl trinitrate
- aspirin 300mg. NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital
- do not routinely give oxygen, only give if sats < 94%*
- perform an ECG as soon as possible but do not delay transfer to hospital. A normal ECG does not exclude ACS

Referral

- current chest pain or chest pain in the last 12 hours with an abnormal ECG: emergency admission
- chest pain 12-72 hours ago: refer to hospital the same-day for assessment
- chest pain > 72 hours ago: perform full assessment with ECG and troponin measurement before deciding upon further action

*NICE suggest the following in terms of oxygen therapy:

- do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:
- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-

92% until blood gas analysis is available.

Patients presenting with stable chest pain

NICE define anginal pain as the following:

- 1. constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arms
- 2. precipitated by physical exertion
- 3. relieved by rest or GTN in about 5 minutes
- patients with all 3 features have typical angina
- patients with 2 of the above features have atypical angina
- patients with 1 or none of the above features have non-anginal chest pain

For patients in whom stable angina cannot be excluded by clinical assessment alone NICE recommend the following (e.g. symptoms consistent with typical/atypical angina OR ECG changes):

- 1st line: CT coronary angiography
- 2nd line: non-invasive functional imaging (looking for reversible myocardial ischaemia)
- 3rd line: invasive coronary angiography

Examples of non-invasive functional imaging:

- myocardial perfusion scintigraphy with single photon emission computed tomography (MPS with SPECT) or
- stress echocardiography or
- first-pass contrast-enhanced magnetic resonance (MR) perfusion or
- MR imaging for stress-induced wall motion abnormalities

+ Q 123

Next question ›

Textbooks

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Links

European Society of Cardiology 👍 14 👎 7

2013 ESC guidelines on the management of stable coronary artery disease

NICE 👍 11 👎 3

Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin

Suggest link Report broken link

Score: **50%**

1 ✗
2 ✓
3 ✗
4 ✗
5 ✓
6 ✓

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Question 7 of 129

A 28 year old woman is admitted to the medical assessment unit complaining of pleuritic chest pain. She is 32 weeks pregnant into her first pregnancy. An ECG reveals a sinus tachycardia. Her blood tests are unremarkable, although the referring accident and emergency doctor added on a D-dimer test for completion and this has come back positive. According to the Royal College of Obstetricians and Gynaecologists guidelines, what is the next suitable investigation for this patient?

- ○ Chest x-ray
- ○ Ventilation/perfusion scan
- ○ Compression duplex doppler of the legs
- ○ CT pulmonary angiogram (CTPA)
- ○ Cardiac catheterisation to assess right sided pressures

Submit answer

Reference ranges

Score: **50%**

1 ✗
2 ✓
3 ✗
4 ✗
5 ✓
6 ✓

7	-

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Question 7 of 129 ✓

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Chest x-ray	52%
Ventilation/perfusion scan	20%
Compression duplex doppler of the legs	22%
CT pulmonary angiogram (CTPA)	6%
Cardiac catheterisation to assess right sided pressures	0%

In suspected pulmonary embolism in pregnant patients the Royal College of Obstetricians and Gynaecologists recommend the following:

- 1. Chest xray to look for an alternative diagnosis.
- 2. If the chest xray is normal consider a compression duplex doppler of both legs to exclude a DVT. If this is positive, the patient is treated with full dose low molecular weight heparin (LMWH) (warfarin is of course teratogenic).
- 3. If both the above investigations are normal, and there remains a strong suspicion of a pulmonary embolism, then clinicians should consider. a CTPA or ventilation perfusion/scan weighing up the risks vs benefits of each and also the local hospital protocol. Current guidance however favours a perfusion scan as it has lower lung radiation doses than a CTPA.

Discuss (10) Improve

Next question

Pregnancy: DVT/PE investigation

Guidelines were updated in 2015 by the Royal College of Obstetricians.

For patients with a suspected deep vein thrombosis (DVT):

- Compression duplex ultrasound should be undertaken where there is clinical suspicion of DVT

For patients with a suspected pulmonary embolism (PE):

- ECG and chest x-ray should be performed in all patients
- In women who also have symptoms and signs of DVT, compression duplex ultrasound should be performed. If compression ultrasonography confirms the presence of DVT, no further investigation is necessary and treatment for VTE should continue
- the decision to perform a V/Q or CTPA should be taken at a local level after discussion with the patient and radiologist

Comparing CTPA to V/Q scanning in pregnancy

CTPA	**V/Q scanning**
CTPA slightly increases the lifetime risk of **maternal breast cancer** (increased by up to 13.6%, background risk of 1/200 for study population). Pregnancy makes breast tissue particularly sensitive to the effects of radiation	V/Q scanning carries a slightly increased risk of **childhood cancer** compared with CTPA (1/50,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it often raised in pregnancy.

AI + Q 123

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RCOG 👍 2 👎 0

Thromboembolic disease in pregnancy and the puerperium: acute management

Suggest link Report broken link

Score: **57.1%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓

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Question 8 of 129

A 54-year-old male previously treated for lymphoma is seen in clinic with a 6-month history of exertional dyspnoea which is progressive. He was treated four years ago and was told that there was no evidence of disease on the final CT scan. His GP treated him for a presumed lower respiratory tract infection two weeks ago. He returned from a business trip to Thailand six months ago, during which he consumed more alcohol than he usually does. He takes no medications and is otherwise fit and well, and is a non-smoker. On examination there is mild pedal oedema, his chest is clear and jugular venous pressure(JVP) is raised on inspiration. Auscultation of his heart reveals an extra heart sound very soon after S2.

What is the likely cause of his symptoms?

- ○ Superior vena cava obstruction
- ○ Cardiac tamponade
- ○ Left ventricular failure
- ○ Chronic obstructive pulmonary disease
- ○ Constrictive pericarditis

Submit answer

Reference ranges

Score: **57.1%**

1 ✗
2 ✓

3	✗
4	✗
5	✓
6	✓
7	✓
8	-

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What is the likely cause of his symptoms?

Superior vena cava obstruction	4%
Cardiac tamponade	5%
Left ventricular failure	24%
Chronic obstructive pulmonary disease	1%
Constrictive pericarditis	65%

Examination of this patient demonstrates Kussmaul's sign - paradoxical elevation of the JVP on inspiration. This sign is seen in constrictive pericarditis, cardiac tamponade and restrictive cardiomyopathy. This presentation gives no other features to suggest tamponade. His constrictive pericarditis here is likely cause by the previous radiotherapy for lymphoma. Other causes of constrictive pericarditis include TB and chronic pericarditis. Definitive management involves surgical pericardial stripping.

👍 👎 Discuss (6) Improve

Next question >

Constrictive pericarditis ★

Causes

- any cause of pericarditis
- particularly TB

Features

- dyspnoea
- right heart failure: elevated JVP, ascites, oedema, hepatomegaly
- JVP shows prominent x and y descent
- pericardial knock - loud S3
- Kussmaul's sign is positive

CXR

- pericardial calcification

The key differences between constrictive pericarditis and cardiac tamponade are summarized in the table below:

	Cardiac tamponade	**Constrictive pericarditis**
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign*	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX *a paradoxical rise in JVP during inspiration

+ Q 123

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Media

PERICARDITIS & PERICARDIAL EFFUSIONS
Osmosis.org

Pericarditis and pericardial effusions

Osmosis - YouTube 👍 5 👎 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 👍 1 👎 0

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Score: **50%**

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3	✗
4	✗
5	✓
6	✓
7	✓
8	✗

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Question 9 of 129

A 38-year-old female presenting with 3 months of increasing abdominal and lower limb swelling. She has a past medical history of myasthenia gravis, associated with a thymic mass. Five years ago, she underwent a thymectomy and after positive margins were noted, she was commenced on a course of radiotherapy. She has no other past medical history. On examination, her heart sounds are quiet with no added sounds. Her chest is clear on auscultation. Her JVP is located at 6 cm above the angle of Louis. Abdominal examination reveals a pulsatile liver and smooth hepatomegaly. A chest x-ray demonstrates only minimal pleural and pericardial calcification. What is the definitive treatment?

- ○ Intravenous furosemide ×
- ○ Intravenous methylprednisolone ×
- ○ Intravenous immunoglobulin ×
- ○ Pericardial drain insertion ×
- ○ Pericardiectomy ×

Submit answer

Reference ranges

Score: **50%**

1 ✗
2 ✓
3 ✗
4 ✗

5 ✓

6 ✓

7 ✓

8 ✗

9 -

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Question 9 of 129 ✓

A 38-year-old female presenting with 3 months of increasing abdominal and lower limb swelling. She has a past medical history of myasthenia gravis, associated with a thymic mass. Five years ago, she underwent a thymectomy and after positive margins were noted, she was commenced on a course of radiotherapy. She has no other past medical history. On examination, her heart sounds are quiet with no added sounds. Her chest is clear on auscultation. Her JVP is located at 6 cm above the angle of Louis. Abdominal examination reveals a pulsatile liver and smooth hepatomegaly. A chest x-ray demonstrates only minimal pleural and pericardial calcification. What is the definitive treatment?

Intravenous furosemide	11%
Intravenous methylprednisolone	10%
Intravenous immunoglobulin	5%
Pericardial drain insertion	14%
Pericardiectomy ✓	60%

The patient presents with clinical features of constrictive pericarditis. Her previous radiotherapy increases the aetiology of radiation-induced pericarditis. The patient has prominent and persistent symptoms for which pericardiectomy is the only definitive treatment available. While diuretics may hold the symptoms prior to surgery or be used in patients not suitable for surgery, pericardiectomy should be the definitive treatment despite the high operative risks. However, radiation induced constrictive pericarditis is known to demonstrate a worse long-term prognosis than other causes.

1. Bertog SC, Thambidorai SK, Parakh K et al. Constrictive pericarditis: etiology and cause-specific survival after pericardiectomy. J Am Coll Cardiol. 2004;43(8):1445.

Discuss (11) Improve

Next question ›

Constrictive pericarditis

Causes
- any cause of pericarditis
- particularly TB

Features
- dyspnoea
- right heart failure: elevated JVP, ascites, oedema, hepatomegaly
- JVP shows prominent x and y descent
- pericardial knock - loud S3
- Kussmaul's sign is positive

CXR
- pericardial calcification

The key differences between constrictive pericarditis and cardiac tamponade are summarized in the table below:

	Cardiac tamponade	**Constrictive pericarditis**
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign*	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX *a paradoxical rise in JVP during inspiration

123

Next question >

Textbooks

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Media

PERICARDITIS &
PERICARDIAL
EFFUSIONS
OSMOSIS.org

Pericarditis and pericardial effusions

Osmosis - YouTube 5 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 1 0

Report broken media

Score: **55.6%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓

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A 76-year-old man with diabetes, hypertension, rheumatoid arthritis and gout presents with a 3 month history of deterioration. He is generally lethargic with reduced energy and has changed from someone who was walking with sticks to now using a frame. He occasionally vomits and has sweating at night time. His wife is concerned that he has lost weight.

On examination, he has clubbing present and a red macular rash across his hands. He complains of his fingers itching and in the abdomen, the spleen is palpable three centimeters below the costal margin. He has a pan systolic murmur and there are crepitations in both lungs. He starts to shake vigorously but remains awake and feels clammy. He is tender across his gluteal muscles.

Hb	109 g/l	Na^+	130 mmol/l
Platelets	276 * 10^9/l	K^+	3.7 mmol/l
WBC	10.4 * 10^9/l	Urea	4.5 mmol/l
Neuts	9.8 * 10^9/l	Creatinine	89 µmol/l
Lymphs	0.4 * 10^9/l	CRP	42 mg/l
Eosin	0.1 * 10^9/l	ESR	32 mm/hr

Blood film	some microcytosis, schistocytes and cell fragments seen
ECG	sinus tachycardia rate 102/min
Chest x-ray	globular heart, bilateral interstitial shadowing

What is the likely diagnosis?

- ○ Lymphoma
- ○ Infective endocarditis
- ○ Congestive cardiac failure
- ○ *Mycoplasma* pneumonia

○ Polymyalgia rheumatica ×

Submit answer

Reference ranges ﹀

Score: **55.6%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	-

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Blood film	some microcytosis, schistocytes and cell fragments seen
ECG	sinus tachycardia rate 102/min
Chest x-ray	globular heart, bilateral interstitial shadowing

What is the likely diagnosis?

Lymphoma	14%
Infective endocarditis	61%
Congestive cardiac failure	5%
Mycoplasma pneumonia	15%

Polymyalgia rheumatica 5%

Strep endocarditis presents insidiously with new murmur

Important for me Less important

This is a chronic inflammatory condition and it can be hard to tell apart the options. Congestive cardiac failure usually has a history of myocardial infarctions and an elevated JVP or ankle oedema which is not seen here. Lymphoma has palpable (cervical, axillary, inguinal) or radiological (mediastinal) evidence of lymphadenopathy although there is splenomegaly here. Polymyalgia rheumatica can present in unusual forms like this but we should look at the other options. *Mycoplasma* can give erythema multiforme which is a macular rash and there is hyponatraemia too. However, the history is too chronic for *mycoplasma* and the unexplained murmur, fevers and raised inflammatory markers should prompt thoughts of a *streptococcus* endocarditis.

👍 👎 Discuss (12) Improve

Next question >

Infective endocarditis: aetiology ★

The strongest risk factor for developing infective endocarditis is a previous episode of endocarditis. The following types of patients are affected:

- previously normal valves (50%, typically acute presentation)
    - the mitral valve is most commonly affected
- rheumatic valve disease (30%)
- prosthetic valves
- congenital heart defects
- intravenous drug users (IVDUs)
    - e.g. typically causing tricuspid lesion)
- others: recent piercings

Causes

- *Staphylococcus aureus*
    - now the most common cause of infective endocarditis
    - particularly common in acute presentation and IVDUs
- *Streptococcus viridans*
    - historically *Streptococcus viridans* was the most common cause of infective endocarditis. This is no longer the case, except in developing countries
    - technically *Streptococcus viridans* is a pseudotaxonomic term, referring to viridans streptococci, rather than a particular organism. The two most notable viridans streptococci are *Streptococcus mitis* and *Streptococcus sanguinis*
    - they are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure
- coagulase-negative Staphylococci such as *Staphylococcus epidermidis*
    - commonly colonize indwelling lines and are the most cause of endocarditis in patients following prosthetic valve surgery, usually the result of perioperative contamination.
    - after 2 months the spectrum of organisms which cause endocarditis return to normal (i.e. *Staphylococcus aureus* is the most common cause)
- *Streptococcus bovis*
    - associated with colorectal cancer
    - the subtype *Streptococcus gallolyticus* is most linked with colorectal cancer
- non-infective
    - systemic lupus erythematosus (Libman-Sacks)
    - malignancy: marantic endocarditis

Culture negative causes

- prior antibiotic therapy
- *Coxiella burnetii*
- *Bartonella*
- *Brucella*
- HACEK: *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)

+ Q 123

Next question ›

Textbooks

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Links

NICE 👍 12 👎 18

2008 Prophylaxis against infective endocarditis

American Heart Association 👍 13 👎 7

Infective endocarditis guidelines

Suggest link

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Media

Endocarditis

Osmosis - YouTube 👍 1 👎 0

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
JANEWAY LESIONS
OSLER NODES
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 0 👎 1

Report broken media

Score: **50%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗

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Question 11 of 129

A 23-year-old woman is referred by the emergency department with sudden onset shortness of breath and pleuritic chest pain. She describes no cough or sputum. The pain is constant and not worse on movement. She is 24 weeks pregnant with her third child. She is normally fit and well and on no regular medication beyond her pregnancy supplements. On examination, she is slightly tachypnoeic at 21/min but her sats are 98% on room air. Her blood pressure is 110/78 mmHg and her heart rate is 86/min. Her chest is clear and the JVP is not elevated and her heart sounds are normal. Examination of her right leg reveals a swelling from above the right knee down to the ankle. There is pitting oedema on the right side as well as collateral superficial veins.

The emergency department registrar has sent several blood tests. The results are:

Hb	118 g/l
Platelets	443 * 10^9/l
WBC	9.8 * 10^9/l

Trop T	6ng/L (<14ng/L excludes cardiac damage)
D-dimer	1.3 mg/L (<0.5)
CRP 16	mg/L (<10)

Her chest x-ray is unremarkable.

Her ABG shows:

pH	7.39
pO2	8.6kPa
pCO2	2.9kPa
HCO3-	21mmol/L (22-29)
Sats	97%
BE	1.1

She is seen on the post take ward round and the Consultant would like to exclude a pulmonary embolism.

What is the most appropriate first investigation for this lady?

- ○ Half dose CTPA ×
- ○ V/Q scan ×
- ○ USS Doppler of legs ×
- ○ Full dose CTPA ×
- ○ No need for investigation treat with LMWH ×

Submit answer

Reference ranges ∨

Score: **50%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	-

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HCO3-	21mmol/L (22-29)
Sats	97%
BE	1.1

She is seen on the post take ward round and the Consultant would like to exclude a pulmonary embolism.

What is the most appropriate first investigation for this lady?

Half dose CTPA	2%
V/Q scan	24%
USS Doppler of legs	62%
Full dose CTPA	6%
No need for investigation treat with LMWH	5%

This lady could well have a PE. However, she also has signs of a deep vein thrombosis in the right leg.

The current guidelines in these situations recommend a CXR followed by USS Doppler of the legs. If a DVT is found they are anticoagulated without any radiation exposure to the baby. If the USS is negative and CXR normal they should then be offered the informed choice of V/Q scan or CTPA. V/Q carries a slightly higher risk to the baby of childhood cancers whilst a CTPA carries a slightly increased lifetime risk of breast cancer to the mother.

Discuss (8) Improve

Next question

Pregnancy: DVT/PE investigation

Guidelines were updated in 2015 by the Royal College of Obstetricians.

For patients with a suspected deep vein thrombosis (DVT):
- Compression duplex ultrasound should be undertaken where there is clinical suspicion of DVT

For patients with a suspected pulmonary embolism (PE):

- ECG and chest x-ray should be performed in all patients
- In women who also have symptoms and signs of DVT, compression duplex ultrasound should be performed. If compression ultrasonography confirms the presence of DVT, no further investigation is necessary and treatment for VTE should continue
- the decision to perform a V/Q or CTPA should be taken at a local level after discussion with the patient and radiologist

Comparing CTPA to V/Q scanning in pregnancy

CTPA	V/Q scanning
CTPA slightly increases the lifetime risk of **maternal breast cancer** (increased by up to 13.6%, background risk of 1/200 for study population). Pregnancy makes breast tissue particularly sensitive to the effects of radiation	V/Q scanning carries a slightly increased risk of **childhood cancer** compared with CTPA (1/50,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it often raised in pregnancy.

+ Q 123

Next question

Textbooks

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Links

RCOG 👍 2 👎 0

Thromboembolic disease in pregnancy and the puerperium: acute management

Suggest link Report broken link

Score: **54.5%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓

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A 22-year-old male presents to clinic complaining of several episodes of heart palpitations. He states that during the attacks his heart beats extremely fast and he feels dizzy.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	95ms
QRS duration	105ms
Events	One episode of tachycardia of 145 bpm with normal QRS morphology and QRS 110ms

What is the most likely diagnosis?

○ Lown-Ganong-Levine syndrome

○ Wolff-Parkinson-White syndrome type A

○ Wolff-Parkinson-White syndrome type B

○ Monomorphic ventricular tachycardia

○ Polymorphic ventricular tachycardia

Submit answer

Reference ranges

Score: **54.5%**

1 ✗

2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	-

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You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	95ms
QRS duration	105ms
Events	One episode of tachycardia of 145 bpm with normal QRS morphology and QRS 110ms

What is the most likely diagnosis?

Lown-Ganong-Levine syndrome	42%
Wolff-Parkinson-White syndrome type A	31%
Wolff-Parkinson-White syndrome type B	11%
Monomorphic ventricular tachycardia	13%
Polymorphic ventricular tachycardia	2%

Lown-Ganong-Levine syndrome (LGL) is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width

Important for me Less important

The key thing to note is the short PR interval. The normal PR interval is 120-200ms. A short PR interval is suggestive of a pre-excitation syndrome. Pre-excitation syndromes occur due to an accessory pathway between the atria with the ventricles, resulting in early depolarisation of the ventricles and a tendency for tachyarrhythmias. Pre-excitation through the accessory pathway results in a short PR interval.

The normal QRS width (normal width <120ms) precludes the diagnosis of Wolff-Parkinson-White syndrome which is characterised by a wide QRS with a slurred upstroke (delta wave). The normal QRS also rules out ventricular tachycardia.

The diagnosis is therefore Lown-Ganong-Levine syndrome (LGL) which is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width.

Discuss (6) | Improve

Next question >

Lown-Ganong-Levine syndrome

Lown-Ganong-Levine (LGL) syndrome is a pre-excitation disorder of the heart which occurs due to an abnormal connection between the atria and ventricles. Similarly, to Wolff-Parkinson-White (WPW) syndrome, the abnormal connection bypasses the AV node. Both of these syndromes can cause episodes of tachycardia which are classed as atrioventricular reentrant tachycardia (AVRT).

Patients with LGL syndrome are prone to episodes of supraventricular tachyarrhythmias (SVT).

Pathophysiology

- Accessory pathway (bundle of James) connects the atria directly to the bundle of His
- This abnormal pathway predisposes to reentrant circuits resulting in episodes of tachyarrhythmias
- Early activation results in pre-excitation resulting in the characteristic short PR interval
- In contrast to WPW syndrome, the bundle of James does not directly activate the myocardium, therefore LGL is characterised by normal QRS morphology and absence of a delta wave

Clinical features

- Episodes of tachyarrhythmias: Palpitations, pre-syncope, syncope, chest pain, shortness of breath

Investigations
- ECG: Short PR interval (<120ms) with normal QRS complex morphology, and absence of a delta wave
- Holter monitor: frequency and severity of attacks
- Electrophysiological studies
- Blood tests: electrolytes and thyroid function
- Echo: To assess for structural heart disease

Management of episodes of SVT
- Vagal manoeuvres
- Adenosine
- Synchronised DC cardioversion if adverse features (as per resuscitation council algorithm)

Long-term management
- Lifestyle modifications: limit alcohol and coffee
- Pharmacological: drugs which decrease AV node conduction may be of use in preventing attacks e.g. beta blockers and rate limiting calcium blockers
- Electrophysiological study and ablation
- Pacemaker: Dual AV sequential demand pacemakers are used where there is an enhanced AV node pathway

AI + Q 123

Next question >

B *I*

Textbooks

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Score: **58.3%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓

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Question 13 of 129

A 60 year old man with a known history of congestive cardiac failure and asthma is reviewed in a cardiology clinic. He is noted to have a blood pressure of 95/63mmHg and a heart rate of 98bpm. An ECG confirms sinus rhythm. He has previously developed symptoms of wheeze with beta blockade. He is commenced on ivabradine 5mg twice daily by his cardiologist.

Which of the following should the patient be warned of as a recognised side effect of ivabradine?

- ○ Neutrophilia
- ○ Hypotension
- ○ Phosphenes
- ○ Diaphoresis
- ○ Renal failure

Submit answer

Reference ranges

Score: **58.3%**

1 ✗
2 ✓
3 ✗
4 ✗
5 ✓

6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	-

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← Question 13 of 129 ✗ →

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Which of the following should the patient be warned of as a recognised side effect of ivabradine?

Neutrophilia	9%
Hypotension	34%
Phosphenes	38%
Diaphoresis	15%
Renal failure	4%

Ivabradine use may be associated with visual disturbances including phosphenes and green luminescence

Important for me Less important

Ivabradine is a second line agent for rate control in the treatment of chronic heart failure in those in whom beta-blockade is contraindicated, not tolerated or therapy fails. Other rate limiting drugs such as the calcium channel blockers diltiazem or verapamil may sometimes be used but hypotension often limits their use. Digoxin too can sometimes be used, even in sinus rhythm although the rate limiting effect it has is relatively modest; often digoxin and ivabradine are ultimately used in conjunction.

NICE guidelines on the use of ivabradine give clear criteria on its use; the patient must have at least a moderate degree of heart failure with a left ventricular ejection fracture of <35%, they must be stable on optimal doses of other heart failure medications including angiotensin system modulators, an aldosterone receptor antagonist and a beta blocker if not contraindicated. It is important the patient is regularly monitored by a community heart failure team to titrate the dose. Ivabradine can also only be used in patients in sinus rhythm. It works by selectively inhibiting the If [ionic funny] channel which is a sodium/potassium symporter channel largely expressed within the sinoatrial node. Inhibition of this channel slows the intrinsic rhythmicity of the hearts pacemaker function. Pharmaceutical bradycardia aids in the management of cardiac failure as it allows improved diastolic filling and reduced myocardial oxygen usage.

Side effects of ivabradine include bradycardia, ventricular escape rhythms, dizziness, headache, muscle cramps and eosinophilia. Neutrophilia is rarely seen with ivabradine. Hypotension is also not seen with ivabradine use which makes it an important drug in the arsenal when treating patients with cardiac failure in whom other rate limiting drugs may be relatively contraindicated.

An important side effect with ivabradine however is that of visual disturbance including the phenomenon of phosphenes, or 'flashing lights'. Other visual symptoms described include green discolouration of visual field, blurring of vision and scintillating scotomata. These visual symptoms are usually transient and mild. They arise due to inhibition of similar ionic channels in the retina to the sinoatrial If channels. Approximately 20% of patients taking the drug develop some form of visual disturbance but only 1% need to discontinue therapy because of it.

The use of ivabradine in renal dysfunction is not well established however manufacturers recommend it may be used with estimated glomerular filtration rates above 15ml/min. There is no evidence its use causes renal dysfunction although mild rises in plasma creatinine levels may be seen.

👍 👎 Discuss (4) Improve

Next question >

Ivabradine

Ivabradine is a class of anti-anginal drug which works by reducing the heart rate. It acts on the I_f ('funny') ion current which is highly expressed in the sinoatrial node, reducing cardiac pacemaker activity.

Adverse effects
- visual effects, particular luminous phenomena, are common
- headache
- bradycardia, heart block

There is no evidence currently of superiority over existing treatments of stable angina.

Next question

Textbooks

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Score: **53.8%**

1 ✗
2 ✓
3 ✗
4 ✗
5 ✓
6 ✓

7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✗

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Question 14 of 129

A 44-year-old gentleman is admitted to hospital with a 2 week history of flu-like symptoms, wheeze, shortness of breath on lying flat and palpitations. There is no background history of note. His observations include a heart rate of 98/min, blood pressure of 96/50 mmHg and saturations of 96% while on 2L of oxygen. He is apyrexial.

On examination you note a low volume pulse with a raised JVP and a raised Y decent. A third heart sound is audible. There are no murmurs. There are bilateral crepitations and pitting oedema of the legs.

Blood investigations indicate:

Hb	158 g/L	Male: (135-180) Female: (115 - 160)
Platelets	335 * 10^9/L	(150 - 400)
WBC	11.7 * 10^9/L	(4.0 - 11.0)
Neuts	8.0 * 10^9/L	(2.0 - 7.0)
Lymphs	2.3 * 10^9/L	(1.0 - 3.5)
Mono	0.7 * 10^9/L	(0.2 - 0.8)
Eosin	0.69 * 10^9/L	(0.0 - 0.4)

Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Three blood cultures have been sent and the preliminary report is negative.

The ECG on admission shows sinus rhythm with left ventricular hypertrophy pattern but no features of ischaemia; the PR interval is normal.

An echocardiogram is performed and notes shows preserved systolic function with moderate concentric left ventricular hypertrophy. Both atria are dilated and there is evidence of diastolic dysfunction. There is no valvular pathology and no suspicious lesions surrounding the valves.

Given this scenario, which imaging modality would be most appropriate in identifying the diagnosis of this patient?

- ○ Transoesophageal echocardiography ×
- ○ Right and left cardiac catheterisation ×
- ○ Myocardial perfusion scan ×
- ○ Cardiac MRI ×
- ○ High resolution CT thorax ×

Submit answer

Reference ranges

Score: **53.8%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓

13	✕
14	-

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Given this scenario, which imaging modality would be most appropriate in identifying the diagnosis of this patient?

Transoesophageal echocardiography	17%
Right and left cardiac catheterisation	14%
Myocardial perfusion scan	6%
Cardiac MRI	58%
High resolution CT thorax	6%

Cardiac MRI is the investigation of choice alongside echocardiography in order to identify the potential cause of cardiomyopathy

Important for me Less important

The echocardiography and examination findings suggest the potential of restrictive cardiomyopathy. In addition this gentleman has significant eosinophilia which could alert us to the possibility of Loffler's endocarditis.

The options provided cover a wide spectrum of cardiac imaging modalities, however cardiac MRI yields the greatest detail with regards to function and structure of the heart in such scenarios.

The possibility of infective endocarditis remains low and a transoesophageal echocardiogram would not necessarily add anything further with regards to the diagnosis.

A catheterisation or myocardial perfusion scan will provide excellent information regarding ischaemia however the information regarding the cause of this restrictive cardiomyopathy will not be revealed.

There is no indication for a high resolution CT thorax given the high grade of suspicion regarding a cardiac primary problem.

👍 👎 Discuss (3) Improve

Next question >

Restrictive cardiomyopathy ★

Causes

- amyloidosis (e.g. secondary to myeloma) - most common cause in UK
- haemochromatosis
- post-radiation fibrosis
- Loffler's syndrome: endomyocardial fibrosis with a prominent eosinophilic infiltrate
- endocardial fibroelastosis: thick fibroelastic tissue forms in the endocardium; most commonly seen in young children
- sarcoidosis
- scleroderma

Pathophysiology

- primarily characterized by decreased compliance of the ventricular endomyocardium
- causes predominately diastolic dysfunction

Features

- similar to constrictive pericarditis
- low-voltage ECG

Features suggesting restrictive cardiomyopathy rather than constrictive pericarditis

- prominent apical pulse
- absence of pericardial calcification on CXR
- the heart may be enlarged
- ECG abnormalities e.g. bundle branch block, Q waves

Investigations

- echocardiography
- cardiac MRI

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

Restrictive cardiomyopathy

Osmosis - YouTube 👍 3 👎 0

CARDIOLOGY
CARDIOMYOPATHY
OVERVIEW

Cardiomyopathy Overview - types (dilated, hypertrophic, restrictive), pathophysiology and treatment

Armando Hasudungan - YouTube 👍 1 👎 0

Report broken media

Score: **50%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
10	✗
11	✓
12	✓
13	✗
14	✗

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A 53-year-old man with a history of diabetes presents to the Emergency Department with crushing central chest pain, vomiting, and diaphoresis.

Observations :

- Pulse: 32 beats per minute.
- Blood pressure: 70/40 mmHg.
- Oxygen saturation: 88%

Examination reveals pale and cool skin. An ECG shows ST-elevation in leads II, III, and aVF, along with complete heart block, and his troponin test is positive. Following initial management, including percutaneous coronary intervention (PCI) with stenting, he is admitted to the intensive care unit.

Three days post admission, he develops severe dyspnoea, cold extremities, and diaphoresis. Auscultation reveals a holosystolic murmur on the left sternal edge. His observations:

- Pulse: 123 per minute
- Blood pressure: 65/45 mmHg.
- Oxygen saturation: 80%

What is the most appropriate next step in management?

○ CT coronary angiography

○ Cardiac MRI

○ Immediate surgical consultation

○ Re-transfer to the catheter lab

○ Transthoracic echocardiography

Submit answer

A 53-year-old man with a history of diabetes presents to the Emergency Department with crushing central chest pain, vomiting, and diaphoresis.

Observations :
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Three days post admission, he develops severe dyspnoea, cold extremities, and diaphoresis. Auscultation reveals a holosystolic murmur on the left sternal edge. His observations:

- Pulse: 123 per minute
- Blood pressure: 65/45 mmHg.
- Oxygen saturation: 80%

What is the most appropriate next step in management?

CT coronary angiography	3%
Cardiac MRI	2%
Immediate surgical consultation	50%
Re-transfer to the catheter lab	8%
Transthoracic echocardiography	37%

Acute heart failure: echocardiography is indicated in new-onset heart failure, cardiogenic shock, suspected valvular or post-MI problems

Important for me Less important

Transthoracic echocardiography is the correct answer. In this scenario, the patient developed cardiogenic shock (hypotension, tachycardia and cool skin) with a holosystolic murmur following a complicated inferior wall STEMI (as denoted by complete heart block on ECG). The differential diagnoses include acute mitral regurgitation (MR) and acute ventricular septal rupture (VSR). A holosystolic murmur heard over the left sternal edge suggests an acute VSR. An urgent transthoracic echocardiogram is warranted to delineate the site and extent of the defect. This diagnostic step should be performed concurrently with medical therapy, preceding an immediate surgical consultation.

CT coronary angiography is not appropriate in this context. Although CT coronary angiography can be useful for patients with suspected stable angina, in this case, where acute VSR is likely following a well-established inferior wall STEMI, echocardiography will provide rapid and precise information about the lesion that will inform surgical management decisions.

Cardiac MRI would not be suitable in this setting. Although it can sometimes provide superior detail compared to echocardiography, its longer scan times, limited availability, and inadequacy for haemodynamically unstable patients render it a less favourable choice for imaging in this emergency.

Immediate surgical consultation plays a pivotal role in managing both acute MR and VSR; however, it should be preceded by a detailed lesion assessment using echocardiography to inform subsequent surgical intervention.

Re-transfer to the catheter lab would not benefit this patient. Such a step may be considered if re-infarction is suspected. Given that the clinical picture points towards an acute VSR post-myocardial infarction, echocardiographic evaluation remains crucial for diagnosis, and coronary angiography will not diagnose such a complication.

Discuss (2) Improve

Next question

Heart failure (acute): features

Acute heart failure (AHF) is a life-threatening emergency. AHF is a term used to describe the sudden onset or worsening of the symptoms of heart failure. Thus it may present with or without a background history of pre-existing heart failure. AHF without a past history of heart failure is called de-novo AHF. Decompensated AHF is more common (66-75%) and presents with a background history of HF.

It usually presents after the age of 65 years and is a major cause of unplanned hospital admission in such patients.

AHF is usually caused by a reduced cardiac output that results from a functional or structural abnormality.

De-novo heart failure is caused by increased cardiac filling pressures and myocardial dysfunction usually as a result of ischaemia. This causes reduced cardiac output and therefore hypoperfusion. This, in turn, can cause pulmonary oedema. Other less common causes of de-novo AHF are:

- Viral myopathy
- Toxins
- Valve dysfunction

Decompensated heart failure accounts for most cases of AHF. The most common precipitating causes of acute AHF are:

- Acute coronary syndrome
- Hypertensive crisis
- Acute arrhythmia
- Valvular disease

There is generally a history of pre-existing cardiomyopathy. It usually presents with signs of fluid congestion, weight gain, orthopnoea and breathlessness.

Patients with heart failure are broadly characterised into 1 of 4 groups based on whether they present:

- With or without hypoperfusion
- With or without fluid congestion

Classifying patients into one of these 4 groups is clinically useful as it determines the therapeutic approach.

Generally speaking, the signs and symptoms of AHF are as follows:

Symptoms	**Signs**
Breathlessness	Cyanosis
Reduced exercise tolerance	Tachycardia
Oedema	Elevated jugular venous pressure
Fatigue	Displaced apex beat
	Chest signs: classically bibasal crackles but may also cause a wheeze
	S3-heart sound

Sometimes the presentation will be that of the underlying cause (e.g: chest pain, viral infection)

Over 90% of patients with AHF have a normal or increased blood pressure (mmHg).

The diagnostic workup for patients with AHF includes:

- Blood tests
    - this is to look for any underlying abnormality such as anaemia, abnormal electrolytes or infection.
- Chest X-ray
    - findings include pulmonary venous congestion, interstitial oedema and cardiomegaly
- Echocardiogram
    - recommended for patients with new-onset heart failure and for patients with known heart failure who have a suspected change in cardiac function

    - particularly important if cardiogenic shock or suspected valvular problems
    - may also identify post-myocardial infarction complications
- B-type natriuretic peptide
    - raised levels (>100mg/litre) indicate myocardial damage and are supportive of the diagnosis.

AI + Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Score: **46.7%**

1 ✗
2 ✓
3 ✗
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15	✗

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Question 16 of 129

A 54-year-old female presents with heart palpitations. She has no past medical history and takes no regular medicines. Her cardiorespiratory exam is normal.

An ECG is performed:

Rate and rhythm	145 beats per minute. Regular rhythm
P waves	Not visible
QRS	145ms. RBBB pattern
QTc	420ms
Axis	Right axis deviation

What is the most likely explanation of these ECG results?

- ○ Ventricular tachycardia ×
- ○ Supraventricular tachycardia with bundle branch block ×
- ○ Torsades de pointes (TdP) ×
- ○ Atrial fibrillation with bundle branch block ×
- ○ Supraventricular tachycardia ×

Submit answer

Reference ranges

Score: **46.7%**

1	✗
2	✓
3	✗
4	✗
5	✓
6	✓
7	✓
8	✗
9	✓
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11	✓
12	✓
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14	✗
15	✗
16	-

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Question 16 of 129 ✗

A 54-year-old female presents with heart palpitations. She has no past medical history and takes no regular medicines. Her cardiorespiratory exam is normal.

An ECG is performed:

Rate and rhythm	145 beats per minute. Regular rhythm
P waves	Not visible
QRS	145ms. RBBB pattern
QTc	420ms
Axis	Right axis deviation

What is the most likely explanation of these ECG results?

Ventricular tachycardia	15%
Supraventricular tachycardia with bundle branch block	61%
Torsades de pointes (TdP)	3%
Atrial fibrillation with bundle branch block	17%
Supraventricular tachycardia	4%

Distinguishing between VT and SVT with BBB can be challenging. The presence of RBBB and RAD favours a diagnosis of SVT with BBB

Important for me Less important

The ECG results confirm a broad complex tachycardia with a regular rhythm. The differential is therefore between supraventricular tachycardia (SVT) with bundle branch block (BBB) and ventricular tachycardia (VT). Distinguishing between VT and SVT with BBB can be challenging. The presence of RBBB and RAD favours a diagnosis of SVT with BBB. If you are unsure of the diagnosis, it is always safer to assume the the patient has VT and treat accordingly.

The absence of P waves should make you consider a diagnosis of atrial fibrillation (AF) with BBB, however the regular rhythm precludes this diagnosis. In SVT, the P waves are often buried within the QRS complex.

The normal QTc and regular rhythm precludes the diagnosis of Torsades de pointes (TdP).

Discuss (7) | Improve

Next question >

Broad complex tachycardia

Features suggesting VT rather than SVT with aberrant conduction

- AV dissociation
- fusion or capture beats
- positive QRS concordance in chest leads
- marked left axis deviation
- history of IHD
- lack of response to adenosine or carotid sinus massage
- QRS > 160 ms

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

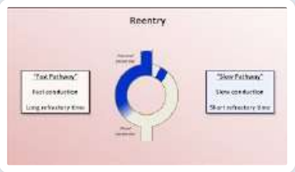
Life in the fast lane 👍 34 👎 7

VT vs SVT

Suggest link

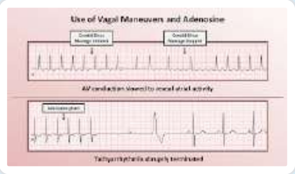
Report broken link

Media


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 0


Use of Vagal Maneuvers and Adenosine

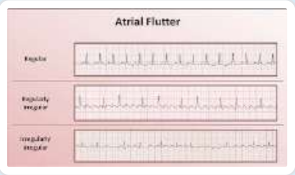
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 2 👎 0

CARDIOLOGY
ARRHYTHMIA

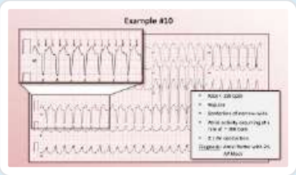
Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 0


Atrial Flutter

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 2 👎 0



Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0

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Score: **43.8%**

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Question 17 of 129

A 39-year-old male is admitted with sharp central chest pain which is worse on inspiration. He states that the pain is partially relieved by leaning forward. He has no past medical history and takes no regular medicines.

ECG results are as follows:

PR segment	Depression
ST segment	Global concave ST elevation

Blood results are as follows:

Troponin	9.82 ug/l (normal range 0.015 - 0.045)

What investigation is most useful diagnostically?

- ○ Cardiac magnetic resonance imaging
- ○ Serial troponins
- ○ Transesophageal echo
- ○ Myocardial Perfusion Imaging (MPI)
- ○ Serial ECGs

Submit answer

Reference ranges

Score: **43.8%**

1	✗
2	✓
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16	✗
17	-

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ECG results are as follows:

PR segment	Depression
ST segment	Global concave ST elevation

Blood results are as follows:

Troponin	9.82 ug/l (normal range 0.015 - 0.045)

What investigation is most useful diagnostically?

Cardiac magnetic resonance imaging	43%
Serial troponins	8%
Transesophageal echo	32%
Myocardial Perfusion Imaging (MPI)	5%
Serial ECGs	12%

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium

Important for me Less important

The patient has clinical and electrocardiographic features of pericarditis. Although a small rise in troponin can be expected in pericarditis, a significant rise should alert you to the possibility of myopericarditis.

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium e.g. regional myocardial oedema, hyperaemia and inflammatory necrosis.

All the other options may be useful, however the most sensitive investigation for diagnosing myopericarditis is cardiac MRI.

👍 | 👎 | 💬 Discuss (6) | Improve

Next question >

Myocarditis ★

Myocarditis describes inflammation of the myocardium. There are a wide range of underlying causes. It should be particularly considered in younger patients who present with chest pain.

Causes
- viral: coxsackie B, HIV
- bacteria: diphtheria, clostridia
- spirochaetes: Lyme disease
- protozoa: Chagas' disease, toxoplasmosis
- autoimmune
- drugs: doxorubicin

Presentation
- usually young patient with an acute history
- chest pain
- dyspnoea
- arrhythmias

Investigations
- bloods
    - ↑ inflammatory markers in 99%
    - ↑ cardiac enzymes
    - ↑ BNP
- ECG
    - tachycardia

    - arrhythmias
    - ST/T wave changes including ST-segment elevation and T wave inversion

Management

- treatment of underlying cause e.g. antibiotics if bacterial cause
- supportive treatment e.g. of heart failure or arrhythmias

Complications

- heart failure
- arrhythmia, possibly leading to sudden death
- dilated cardiomyopathy: usually a late complication

+ Q 123

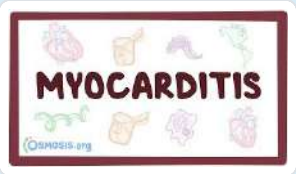
Next question >

Textbooks

High-yield textbook

Extended textbook

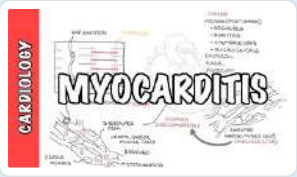
Media



Myocarditis

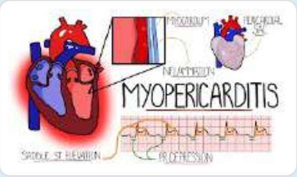
Osmosis - YouTube

8 0


CARDIOLOGY
MYOCARDITIS

Myocarditis

Armando Hasudungan - YouTube 👍 3 👎 0


MYOPERICARDITIS

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 0

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Score: **41.2%**

1	✗
2	✓
3	✗
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12	✓
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15	✗
16	✗

17 ✗

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Question 18 of 129

A 64-year-old man presents to cardiology outpatients with intermittent exertional chest pain. He has suffered from angina for several years and finds his glyceryl trinitrate spray effective, but he is keen on further treatment to help his symptoms.

His past medical history includes angina, asthma, and type 2 diabetes. His current medication includes salbutamol, montelukast, Fostair, metformin, amlodipine, glyceryl trinitrate, aspirin and simvastatin.

Observations are temperature 37ºC, heart rate 52 beats/min, blood pressure 98/59 mmHg, respiratory rate 14 breaths/min, and oxygen saturation 96%.

What is the most appropriate additional therapy?

- ○ Bisoprolol ×
- ○ Isosorbide mononitrate ×
- ○ Ivabradine ×
- ○ Ranolazine ×
- ○ Verapamil ×

Submit answer

Reference ranges

Score: **41.2%**

1 ×

2	✓
3	✗
4	✗
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18	-

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Observations are temperature 37°C, heart rate 52 beats/min, blood pressure 98/59 mmHg, respiratory rate 14 breaths/min, and oxygen saturation 96%.

What is the most appropriate additional therapy?

Bisoprolol	9%
Isosorbide mononitrate	17%
Ivabradine	8%
Ranolazine	52%
Verapamil	13%

For a patient with symptomatic stable angina on a calcium channel blocker but with a contraindication to a beta-blocker, the next line treatment should be long-acting nitrate, ivabradine, nicorandil or ranolazine

Important for me Less important

The correct answer is **ranolazine**. This patient has stable angina despite the use of first-line anti-anginal therapy with amlodipine. NICE recommends using either a beta-blocker or a dihydropyridine calcium channel blocker as first-line anti-anginal.

There are a number of options for next-line treatment for patients on a calcium channel blocker, including the addition of a beta-blocker, long-acting nitrate, ivabradine, nicorandil or ranolazine. In this case, **ranolazine** would be the most appropriate, as the other options have either cautions or contra-indications.

Given that this patient has asthma, it would be best practice to avoid a beta-blocker such as **bisoprolol**, especially if other equally appropriate therapy options are available.

Given that this patient already has a slightly low blood pressure on the candesartan alone, it would be best to avoid adding in **isosorbide mononitrate** as this risks dropping the blood pressure further.

Ivabradine is contra-indicated in bradycardia. The BNF states 'do not initiate for angina if heart rate below 70 beats per minute'.

The patient is already on a dihydropyridine calcium channel blocker, therefore adding a non-dihydropyridine calcium channel blocker such as **verapamil** should be avoided.

👍 👎 Discuss (4) Improve

Next question >

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used

- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 22 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

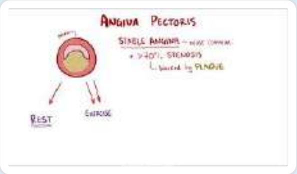
Suggest link
Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **38.9%**

1	✗
2	✓
3	✗
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17	✗
18	✗

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Question 19 of 129

A 35-year-old woman presented to the emergency department with intermittent palpitations, breathlessness, and non-specific chest discomfort. The previous day she had found out that she was 14-weeks pregnant which had caused significant stress and anxiety. She has a long history of anxiety and depression, managed by cognitive behavioural therapy. She reported that she had intermittently had episodes of palpitations and shortness of breath for the past 10 years and had attributed this to her anxiety. These symptoms had worsened over the past couple of weeks and today her symptoms were intolerable. Her medications consisted of over-the-counter vitamin supplements.

She looked anxious, but otherwise, the examination was unremarkable.

An ECG was performed:

ECG #1: Normal sinus rhythm. T wave inversion in V1 - V3.

Whilst in the emergency department, she complained of severe palpitations and dizziness.

Her ECG was repeated:

ECG #2: Wide complex regular tachycardia with left bundle branch block (LBBB) pattern. Heart rate 150bpm. Fusion beats and capture beats present.

As she was being transferred into resus, she spontaneously cardioverted back to sinus rhythm.

An echocardiogram was performed:

The right ventricle is dilated with depressed right ventricular systolic function and localised apical aneurysm. Left ventricular size and function are normal.

What is the most likely cause of her presentation?

○ Aortic dissection ×

○ Takutsubo cardiomyopathy ×

○ Peripartum cardiomyopathy ×

○ Arrhythmogenic right ventricular cardiomyopathy ×

○ Brugada syndrome ×

Submit answer

Reference ranges ∨

Score: **38.9%**

1	✗
2	✓
3	✗
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17	✗

18	✗
19	-

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A 35-year-old woman presented to the emergency department with intermittent palpitations, breathlessness, and non-specific chest discomfort. The previous day she had found out that she was 14-weeks pregnant which had caused significant stress and anxiety. She has a long history of anxiety and depression, managed by cognitive behavioural therapy. She reported that she had intermittently had episodes of palpitations and shortness of breath for the past 10 years and had attributed this to her anxiety. These symptoms had worsened over the past couple of weeks and today her symptoms were intolerable. Her medications consisted of over-the-counter vitamin supplements.

She looked anxious, but otherwise, the examination was unremarkable.

An ECG was performed:

ECG #1: Normal sinus rhythm. T wave inversion in V1 - V3.

Whilst in the emergency department, she complained of severe palpitations and dizziness.

Her ECG was repeated:

ECG #2: Wide complex regular tachycardia with left bundle branch block (LBBB) pattern. Heart rate 150bpm. Fusion beats and capture beats present.

As she was being transferred into resus, she spontaneously cardioverted back to sinus rhythm.

An echocardiogram was performed:

The right ventricle is dilated with depressed right ventricular systolic function and localised apical aneurysm. Left ventricular size and function are normal.

What is the most likely cause of her presentation?

Aortic dissection	1%
Takutsubo cardiomyopathy	35%
Peripartum cardiomyopathy	6%
Arrhythmogenic right ventricular cardiomyopathy	54%
Brugada syndrome	4%

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Important for me Less important

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterised by the replacement of the myocardium of the right ventricle with fibrofatty tissue. The abnormal fibrofatty tissue predisposes to ventricular tachycardia (VT), right ventricular systolic dysfunction, and can cause sudden death. It is often inherited but can be sporadic. Presentation is often with nonspecific palpitations, chest discomfort, and syncope. This patient has a typical presentation: the pregnancy-associated increase in stroke volume is likely the trigger for her symptoms recently worsening. Her two ECGs are characteristic: typical changes include inverted T waves in the precordial leads V1-V3 (looking at the right ventricle) with episodes of ventricular tachycardia originating in the right ventricle (giving the ECG a LBBB morphology as the arrhythmia takes an abnormal route of conduction from right to left through slowly-conducting myocardium rather than conducting tissue). Epsilon waves may also be seen in ARVC. Her echocardiogram is also characteristic - infiltration and thinning of the right ventricular wall can occur to the point that systolic function is impaired and an aneurysm forms.

Note that the differential for a wide-complex regular tachycardia on an ECG includes SVT with aberrant conduction (as well as VT). However, the fusion beats and capture beats are more in keeping with VT in this case.

This patient's presentation is not typical for aortic dissection. An echo might see cardiac tamponade or new aortic regurgitation if this was the diagnosis but this is not the case here.

Takutsubo cardiomyopathy is caused by transient systolic dysfunction of the left ventricle (LV), presenting similarly to an acute coronary syndrome. Patients typically have chest pain, a raised troponin, and ECG findings consistent with ischaemia such as ST elevation/T wave inversion. This is typically triggered by recent physical or emotional stress. Patients are initially managed as an ACS but cardiac angiography shows no significant coronary artery disease. Instead, the LV wall shows hypokinesis or akinesis which creates the shape of an 'octopus pot' during systole. It may be argued that this patient has experienced a recent stressor (the news of pregnancy) however her presentation, long history of symptoms, and echo findings are more in keeping with AVRC.

Peripartum cardiomyopathy (PPCM) is a rare type of cardiomyopathy that is often dilated and occurs in late pregnancy or early postpartum (typically one month before or after birth). The aetiology is unclear but is thought to be multifactorial. Criteria for diagnosis of PPCM include impairment of LV systolic function (LVEF <45%) which was not the case in this patient. Furthermore, this patient presented in the early second trimester - too early for PPCM but late enough for pregnancy-related haemodynamic changes to trigger her underlying ARVC.

Lastly, Brugada syndrome is another important differential to consider in young patients with palpitations, ECG changes in V1-V3, and episodes of VT. Brugada is caused by an inherited mutation in the cardiac sodium channel. However, echocardiography is typically normal and an ECG may show unusual ST elevation in V1-V3 with T wave inversion in these leads.

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Next question >

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology

- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation

- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

123

Next question

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Links

Life in the fast lane 👍 13 👎 7

Arrhythmogenic right ventricular cardiomyopathy ECG

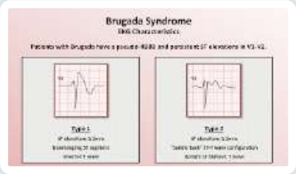
Suggest link Report broken link

Media

CARDIOLOGY
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

Report broken media

Score: **36.8%**

1 ✗
2 ✓

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A 67-year-old woman with recurrent pulmonary emboli secondary to antiphospholipid syndrome presents with worsening dyspnoea on exertion. The dyspnoea is accompanied by chest pain when she climbs the stairs.

On examination, she is on 2L oxygen and saturating at 94%, there is cyanosis seen under the tongue, a raised jugular venous pressure is seen and a right ventricular heave is felt.

Troponin	3 ng/L	(<14)
Pro-Brain Natriuretic Peptide	2300 pg/ml	(<100)

The CT pulmonary angiogram does not demonstrate any large pulmonary emboli. An echocardiogram shows right ventricle hypertrophy and moderate tricuspid regurgitation. Upon right heart catheterisation, the pressures in the pulmonary artery are 30mmHg and after inhaled nitric oxide drops to 20mmHg.

What is the next best management option?

○ Bosentan

○ Iloprost

○ Increase apixaban dose

○ Nifedipine

○ Sildenafil

Submit answer

Reference ranges

A 67-year-old woman with recurrent pulmonary emboli secondary to antiphospholipid syndrome presents with worsening dyspnoea on exertion. The dyspnoea is accompanied by chest pain when she climbs the stairs.

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The CT pulmonary angiogram does not demonstrate any large pulmonary emboli. An echocardiogram shows right ventricle hypertrophy and moderate tricuspid regurgitation. Upon right heart catheterisation, the pressures in the pulmonary artery are 30mmHg and after inhaled nitric oxide drops to 20mmHg.

What is the next best management option?

Bosentan	12%
Iloprost	6%
Increase apixaban dose	1%
Nifedipine	73%
Sildenafil	8%

Pulmonary arterial hypertension patients with positive response to vasodilator testing should be treated with calcium channel blockers

Important for me Less important

Nifedipine is the correct option as an oral calcium channel blocker to treat this vasodilator (nitric oxide) responsive form of pulmonary arterial hypertension. The calcium channel blocker has proven efficacious in dilating the pulmonary vasculature to a sufficient degree to improve her symptoms.

Bosentan is an incorrect option as it is an endothelin receptor antagonist, which is used in patients who do not respond to vasodilator testing.

Iloprost is an incorrect option as it is a prostacyclin analogue, best utilised when the patient has not responded to vasodilator testing.

Sildenafil is an incorrect option as this would be a viable treatment option for the majority of patients because the majority do not respond to vasodilator testing. Sildenafil is a phosphodiesterase type 5 inhibitor.

Increase apixaban dose when the patient is on the recommended dose is an incorrect option because this will be an unlicensed use of the apixaban and would not reverse pulmonary hypertension which has likely been established from previous, recurrent pulmonary emboli.

👍 👎 💬 Discuss (4) Improve

Next question >

Pulmonary arterial hypertension ★

Pulmonary arterial hypertension (PAH) may be defined as a resting mean pulmonary artery pressure of >= 20 mmHg. Endothelin is thought to play a key role in the pathogenesis of PAH. It is more common in females and typically presents between the ages of 30-50 years. Pulmonary hypertension may of course develop secondary to chronic lung diseases such as COPD - PAH is diagnosed in the absence of this although certain factors increase the risk, including HIV, cocaine and anorexigens (e.g. fenfluramine). Around 10% of cases are inherited in an autosomal dominant fashion.

Features

- progressive exertional dyspnoea is the classical presentation
- other possible features include exertional syncope, exertional chest pain and peripheral oedema
- cyanosis
- clinical signs:
  - right ventricular heave: indicating right ventricular hypertrophy or dilatation
  - loud P2: early in the disease reflects increased pulmonary artery pressure and may be accompanied by a palpable P2 in severe cases. With advanced PAH there may be right ventricular failure leading to a soft S2
  - raised JVP with prominent 'a' waves: reflects increased resistance to right atrial emptying due to elevated right ventricular end-diastolic pressure
  - tricuspid regurgitation

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that **acute vasodilator testing** is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide.

If there is a positive response to acute vasodilator testing (a minority of patients)

- oral calcium channel blockers

If there is a negative response to acute vasodilator testing (the vast majority of patients)

- prostacyclin analogues: treprostinil, iloprost
- endothelin receptor antagonists
  - non-selective: bosentan
  - selective antagonist of endothelin receptor A: ambrisentan
- phosphodiesterase inhibitors: sildenafil

Patients with progressive symptoms should be considered for a heart-lung transplant.

Next question >

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Links

European Society of Cardiology 👍 12 👎 13

2015 Guidelines for the diagnosis and treatment of pulmonary hypertension

Medcomic 👍 14 👎 8

Pulmonary hypertension

Respiratory Medicine journal 👍 13 👎 7

Pulmonary vasodilator testing and use of calcium channel blockers in pulmonary arterial hypertension

Suggest link

Report broken link

Media

Pulmonary hypertension

Osmosis - YouTube 👍 1 👎 4

Report broken media

Score: **35%**

1	✗
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Question 21 of 129

A 55-year-old man presents to the emergency department with sudden cardiac arrest. The patient has no known history of heart disease. The initial pulse and rhythm analysis confirms pulseless ventricular tachycardia.

What medications are indicated as part of the advanced life support algorithm?

- ○ Adrenaline after third shock
- ○ Adrenaline and amiodarone after third shock
- ○ Adrenaline and amiodarone immediately
- ○ Adrenaline immediately
- ○ None

Submit answer

Reference ranges

Score: **35%**

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21	-

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A 55-year-old man presents to the emergency department with sudden cardiac arrest. The patient has no known history of heart disease. The initial pulse and rhythm analysis confirms pulseless ventricular tachycardia.

What medications are indicated as part of the advanced life support algorithm?

Adrenaline after third shock	17%
Adrenaline and amiodarone after third shock	47%
Adrenaline and amiodarone immediately	3%
Adrenaline immediately	27%
None	6%

In ALS, amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.

Important for me Less important

This patient has a shockable rhythm (pulseless ventricular tachycardia (VT)) and therefore requires adrenaline and amiodarone to be administered after the third cycle.

Adrenaline after the third cycle is incorrect; this should be given with amiodarone for shockable rhythms.

As per the resuscitation guidelines, giving **adrenaline + amiodarone immediately** is incorrect as these should only be given after three cycles.

Adrenaline immediately is incorrect as this is the treatment for non-shockable rhythms, however pulseless VT is considered a shockable rhythm.

To give **none** is incorrect as the resuscitation guidelines state that both adrenaline and amiodarone have a role in shockable cardiac arrests.

👍 👎 Discuss (4) Improve

Next question >

Adult advanced life support ★

The following is based on the 2021 Resus Council guidelines. Please see the link for more details, below is only a very brief summary of key points.

It should be remembered that the algorithm divides patients into those with:
- 'shockable' rhythms: ventricular fibrillation/pulseless ventricular tachycardia (VF/pulseless VT)
- 'non-shockable' rhythms: asystole/pulseless-electrical activity (asystole/PEA)

Major points include:
- chest compressions
  - the ratio of chest compressions to ventilation is 30:2
  - chest compressions are now continued while a defibrillator is charged
- defibrillation
  - a single shock for VF/pulseless VT followed by 2 minutes of CPR
  - if the cardiac arrested is witnessed in a monitored patient (e.g. in a coronary care unit) then the 2015 guidelines recommend 'up to three quick successive (stacked) shocks', rather than 1 shock followed by CPR
- drug delivery
  - IV access should be attempted and is first-line
  - if IV access cannot be achieved then drugs should be given via the intraosseous route (IO)
  - delivery of drugs via a tracheal tube is no longer recommended
- adrenaline
  - adrenaline 1 mg as soon as possible for non-shockable rhythms
  - during a VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock

    - repeat adrenaline 1mg every 3-5 minutes whilst ALS continues
- amiodarone
    - amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.
    - a further dose of amiodarone 150 mg should be given to patients who are in VF/pulseless VT after 5 shocks have been administered
    - lidocaine used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead
- thrombolytic drugs
    - should be considered if a pulmonary embolus is suspected
    - if given, CPR should be continued for an extended period of 60-90 minutes

Other points

- atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA)
- following successful resuscitation oxygen should be titrated to achieve saturations of 94-98%. This is to address the potential harm caused by hyperoxaemia

Reversible causes of cardiac arrest:

The 'Hs'	The 'Ts'
• Hypoxia • Hypovolaemia • Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders • Hypothermia	• Thrombosis (coronary or pulmonary) • Tension pneumothorax • Tamponade - cardiac • Toxins

123

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Links

Resuscitation Council 👍 32 👎 14

2021 Basic Life Support guidelines

Resuscitation Council 👍 58 👎 25

2021 Advanced Life Support guidelines

Suggest link

Report broken link

Media

RC (UK) Cardiac Arrest Management Demo

Resuscitation Council (UK) - YouTube 👍 16 👎 2

Report broken media

Score: **33.3%**

1	✗
2	✓
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Question 22 of 129

A 64-year-old man is reviewed on the coronary care unit and is making a good recovery 2 days post-myocardial infarction.

On examination, he appears well and is haemodynamically stable. His heart rate is 72 beats/min and his blood pressure is 132/84 mmHg.

A 12-lead ECG is performed and shows isorhythmic dissociation between P waves and QRS complexes. The QRS complexes are wide and fusion beats are visible on the ECG.

Given the likely diagnosis, which of the following is the most appropriate management?

- ○ Amiodarone
- ○ Atropine
- ○ Discharge with standard follow up
- ○ Magnesium
- ○ Pacemaker insertion

Submit answer

Reference ranges

Score: **33.3%**

1 ✗
2 ✓
3 ✗

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A 12-lead ECG is performed and shows isorhythmic dissociation between P waves and QRS complexes. The QRS complexes are wide and fusion beats are visible on the ECG.

Given the likely diagnosis, which of the following is the most appropriate management?

Amiodarone	15%
Atropine	2%
Discharge with standard follow up	50%
Magnesium	4%
Pacemaker insertion	30%

Accelerated idioventricular rhythm is common and unconcerning following recent MI

Important for me Less important

The diagnosis here is accelerated idioventricular rhythm (AIVR). This is a benign ectopic rhythm of ventricular origin. It usually occurs with reperfusion following a myocardial infarction. It is differentiated from ventricular bradycardias and tachycardia by its normal rate - in this case, 72 beats/min. Reperfusion of ischaemic tissue leads to increased depolarisation rate of ventricular myocytes, which then override the sinoatrial node, leading to dissociation between the atrial and ventricular rhythms as seen here (P waves and QRS are isorhythmically dissociated). As the patient is hemodynamically stable, treatment is not necessary;

AIVR is generally self-limiting.

Amiodarone would be used in the cases of ventricular tachycardia. Although there are some overlapping features on the ECG, the patient's heart rate is normal and AIVR is strongly suggested here. Antiarrhythmics are therefore unnecessary.

Atropine is occasionally used to increase the sinus rate and overcome AIVR, however, this is mainly in symptomatic patients. Generally, the condition is self-limiting and in hemodynamically stable patients no treatment is required.

Magnesium is used to treatment torsades de pointes, a polymorphic form of ventricular tachycardia which is recognisable on ECG. It plays no role in the management of AIVR.

Pacemaker insertion is unwarranted. As described above, this condition is self-limiting.

👍 👎 💬 Discuss (5) Improve

Next question >

Accelerated idioventricular rhythm ★

Accelerated idioventricular rhythm (AIVR) is a benign ectopic rhythm of ventricular origin. It usually occurs following the reperfusion of an ischaemic myocardium. It has a rate of 50-110 beats per minute, which helps differentiate it from ventricular bradycardia or ventricular tachycardia.

Pathophysiology:

- Reperfusion of ischaemic tissue, electrolyte abnormalities or drug toxicity leads to an increased depolarisation rate of ventricular myocytes.
- When this depolarisation rate is faster than the rate produced by the sinoatrial node, it becomes the overriding rhythm. Therefore, sinus bradycardia by vagal excess or reduced sympathetic activity can precipitate AIVR.

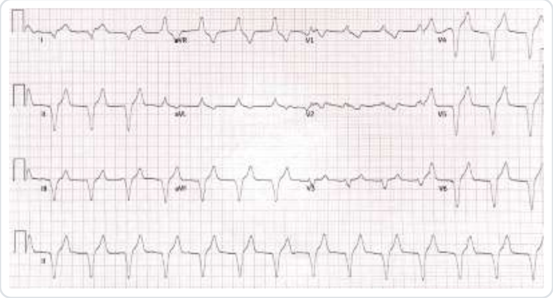
AIVR has a rate of between 50 -110 beats per minute and, therefore, the patient tends to be haemodynamically stable. However, if the rate is fast or there is extensive damage to the myocardium, the patient can become unstable.

Causes:

- Reperfusion following myocardial infarction - by far the most common cause
- Beta-sympathomimetics (e.g. adrenaline)
- Drug toxicity (digoxin/cocaine)
- Electrolyte imbalance
- Cardiomyopathy, congenital heart disease, myocarditis

AIVR is diagnosed based on electrocardiography (ECG) findings:

- AIVR has a gradual onset and termination as the ventricular rate is only slightly faster than sinus rhythm. This may result in ventricular fusion beats on ECG.
- Atrioventricular dissociation - P waves present but not associated with QRS complexes.
- The ECG will show wide QRS complexes >120ms.
- Rate between 50-110 beats per minute.



Treatment:

- AIVR is usually self-limiting and therefore treatment is not necessary, however, occasionally atropine can be used to increase the sinus rate to overcome AIVR.

123

Next question

Textbooks

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Score: **31.8%**

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22 ✗

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Question 23 of 129

You receive a phone call requesting advice from a GP. A patient who is on long term warfarin for atrial fibrillation has been found to have an INR of 10.0 following a recent course of antibiotics. She is not bleeding and only has long standing senile purpura on her arms with no new bruising. What is the most appropriate advice to give?

- ○ Admit for intravenous vitamin K and monitoring
- ○ Give oral vitamin K and continue warfarin at usual dose
- ○ Give oral vitamin K and stop warfarin until INR < 5
- ○ Give oral vitamin K and stop warfarin until INR < 3
- ○ Stop warfarin and restart when INR < 3

Submit answer

Reference ranges

Score: **31.8%**

1	✗
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6	✓
7	✓
8	✗

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Question 23 of 129 ✗

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Admit for intravenous vitamin K and monitoring	16%
Give oral vitamin K and continue warfarin at usual dose	0%
Give oral vitamin K and stop warfarin until INR < 5	65%
Give oral vitamin K and stop warfarin until INR < 3	13%
Stop warfarin and restart when INR < 3	6%

INR > 8.0 (no bleeding) - stop warfarin, give oral vitamin K 1-5mg, repeat dose of vitamin K if INR high after 24 hours, restart when INR < 5.0

Important for me Less important

The correct answer is to **give oral vitamin K and stop warfarin until INR < 5**. This is because the patient has a significantly elevated INR of 10.0 but no active bleeding or high risk of bleeding. According to the UK guidelines, in this situation, it is appropriate to give oral vitamin K (usually 1-5 mg) and withhold warfarin until the INR is below 5. This will allow for a rapid reduction in INR and reduce the risk of bleeding complications.

The option **admit for intravenous vitamin K and monitoring** is not the most appropriate choice in this case, as the patient does not have any active bleeding or high risk of bleeding. Intravenous vitamin K and hospital admission are generally reserved for patients with life-threatening bleeding or an extremely high risk of bleeding.

The option **give oral vitamin K and continue warfarin at usual dose** would not be suitable, as continuing warfarin without interruption would maintain an elevated INR level, increasing the risk of bleeding complications. It is important to withhold warfarin temporarily to allow the INR to decrease.

The option **give oral vitamin K and stop warfarin until INR < 3** might seem reasonable; however, stopping warfarin until an INR of less than 3 could lead to subtherapeutic anticoagulation levels for this patient with atrial fibrillation. The goal therapeutic range for most patients on warfarin with atrial fibrillation is an INR between 2 and 3. Stopping warfarin until the INR falls below this range may increase their thromboembolic risk.

Finally, choosing to simply **stop warfarin and restart when INR < 3**, without administering oral vitamin K, would result in a slower reduction of INR. This could leave the patient at an increased risk of bleeding complications for a longer period. Oral vitamin K helps to lower the INR more rapidly and safely.

👍 👎 Discuss (3) Improve

Next question >

Warfarin: management of high INR ★

The following is based on the BNF guidelines, which in turn take into account the British Committee for Standards in Haematology (BCSH) guidelines.

Situation	Management
Major bleeding (e.g. variceal haemorrhage, intracranial haemorrhage)	Stop warfarin Give intravenous vitamin K 5mg Prothrombin complex concentrate - if not available then FFP*
INR > 8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Repeat dose of vitamin K if INR

Situation	Management
	still too high after 24 hours Restart warfarin when INR < 5.0
INR > 8.0 **No bleeding**	Stop warfarin Give vitamin K 1-5mg by mouth, using the intravenous preparation orally Repeat dose of vitamin K if INR still too high after 24 hours Restart when INR < 5.0
INR 5.0-8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Restart when INR < 5.0
INR 5.0-8.0 **No bleeding**	Withhold 1 or 2 doses of warfarin Reduce subsequent maintenance dose

*as FFP can take time to defrost prothrombin complex concentrate should be considered in cases of intracranial haemorrhage

AI + Q 123

Next question >

Textbooks

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Extended textbook

Links

BNF 👍 16 👎 11

Warfarin

BNF 👍 18 👎 10

Oral anticoagulants

Suggest link

Report broken link

Score: **30.4%**

1	✗
2	✓
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23 ✗

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Question 24 of 129

A 65-year-old man is reviewed in the Emergency Department. Twelve months ago he had a prosthetic aortic valve replacement for progressive aortic stenosis. Since the operation he has been well but over the past two weeks he has been complaining of lethargy, fever and anorexia. Blood tests show a CRP of 215 mg/l. The chest x-ray and urine dip are unremarkable.

Given the suspicion of infective endocarditis an transoesophageal echocardiogram is performed which demonstrates a large vegetative lesion around the prosthetic aortic valve.

What is the most appropriate antibiotic treatment whilst awaiting blood cultures?

- ○ IV amoxicillin ×
- ○ IV teicoplanin + vancomycin ×
- ○ IV flucloxacillin + low-dose gentamicin ×
- ○ IV vancomycin + rifampicin + low-dose gentamicin ×
- ○ IV teicoplanin + rifampicin ×

Submit answer

Reference ranges

Score: **30.4%**

1 ✗
2 ✓

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A 65-year-old man is reviewed in the Emergency Department. Twelve months ago he had a prosthetic aortic valve replacement for progressive aortic stenosis. Since the operation he has been well but over the past two weeks he has been complaining of lethargy, fever and anorexia. Blood tests show a CRP of 215 mg/l. The chest x-ray and urine dip are unremarkable.

Given the suspicion of infective endocarditis an transoesophageal echocardiogram is performed which demonstrates a large vegetative lesion around the prosthetic aortic valve.

What is the most appropriate antibiotic treatment whilst awaiting blood cultures?

IV amoxicillin	3%
IV teicoplanin + vancomycin	2%
IV flucloxacillin + low-dose gentamicin	16%
IV vancomycin + rifampicin + low-dose gentamicin	78%
IV teicoplanin + rifampicin	1%

IV vancomycin + rifampicin + low-dose gentamicin is the empirical treatment of choice in prosthetic valve endocarditis

Important for me Less important

The correct answer is **IV vancomycin + rifampicin + low-dose gentamicin**. In a patient with a prosthetic valve and suspected infective endocarditis, the most likely causative organisms are staphylococci (including methicillin-resistant Staphylococcus aureus [MRSA]), streptococci, and enterococci. The combination of IV vancomycin, rifampicin, and low-dose gentamicin provides coverage against these organisms while awaiting blood culture results. Vancomycin is effective against MRSA and other gram-positive bacteria, rifampicin has good penetration into biofilms on prosthetic material and enhances the activity of vancomycin, and gentamicin provides additional synergistic bactericidal activity.

IV amoxicillin is not the most appropriate choice in this case because it does not provide adequate coverage for MRSA or resistant enterococci. Amoxicillin is typically used as part of empirical treatment for native valve endocarditis caused by streptococci but would not be sufficient for a prosthetic valve infection where staphylococcal involvement is more common.

IV teicoplanin + vancomycin would not be an appropriate choice because combining two glycopeptides offers no additional benefit over using just one of them. Both teicoplanin and vancomycin have similar spectra of activity against gram-positive bacteria; however, they do not cover gram-negative organisms or provide synergy with other antibiotics.

IV flucloxacillin + low-dose gentamicin would be inadequate in this situation as flucloxacillin does not cover MRSA or resistant enterococci. Flucloxacillin is effective against methicillin-sensitive staphylococci but would not provide broad enough coverage for a prosthetic valve infection where resistant organisms are more likely to be involved.

IV teicoplanin + rifampicin is not the best choice because it lacks the synergistic bactericidal activity provided by the addition of low-dose gentamicin. Although teicoplanin and rifampicin would cover most gram-positive organisms, including MRSA, the absence of gentamicin could result in suboptimal treatment for enterococci and other resistant bacteria.

In conclusion, **IV vancomycin + rifampicin + low-dose gentamicin** is the most appropriate empirical antibiotic treatment for a patient with suspected infective endocarditis involving a prosthetic valve. This regimen provides broad coverage against common causative organisms while awaiting blood culture results and can be adjusted based on culture sensitivities once available.

👍 👎 💬 Discuss (5) Improve

Next question >

Infective endocarditis: prognosis and management

Poor prognostic factors
- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism
- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	Suggested antibiotic therapy
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin If prosthetic valve • vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA • vancomycin + rifampicin

Scenario	Suggested antibiotic therapy
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA - vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic - vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic - vancomycin + low-dose gentamicin

Indications for surgery
- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
DUKE'S CRITERIA
JANEWAY LESIONS
OSLER NODES

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **29.2%**

1 ✗
2 ✓
3 ✗

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Question 25 of 129

A 71-year-old patient presents to the Emergency Department with a 30 minute history of crushing central chest pain. ECG shows tall R waves in V1-2. Which coronary territory is likely to be affected?

- ○ Lateral
- ○ Posterior
- ○ Anteroseptal
- ○ Anterolateral
- ○ Inferior

Submit answer

Reference ranges

Score: **29.2%**

1	✗
2	✓
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25	-

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A 71-year-old patient presents to the Emergency Department with a 30 minute history of crushing central chest pain. ECG shows tall R waves in V1-2. Which coronary territory is likely to be affected?

Lateral	3%
Posterior	55%
Anteroseptal	36%
Anterolateral	4%
Inferior	3%

Posterior MI typically present on ECG with tall R waves V1-2

Important for me Less important

Discuss (2) Improve

Next question >

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary

	ECG changes	Coronary artery
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

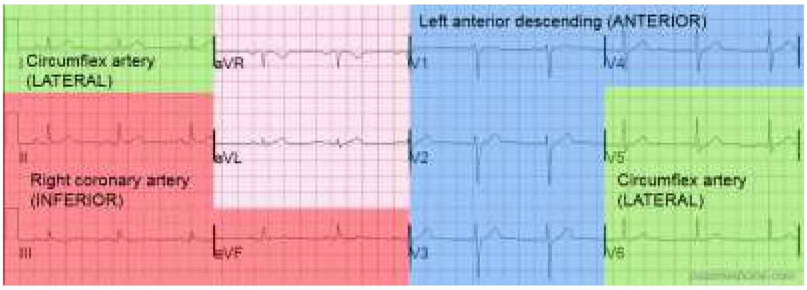
Left anterior descending (ANTERIOR)
Circumflex artery (LATERAL)
aVR
V1
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 7 👎 11

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 2 👎 6

Classic circumflex infarction ECG

Suggest link

Report broken link

Score: **28%**

1	✗
2	✓
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Question 26 of 129

A 64-year-old man with mitral regurgitation attends the cardiology clinic for review. An ECG reveals new atrial fibrillation.

He is well and denies any shortness of breath or chest pain. His blood pressure is 115/98 mmHg and his heart rate is 64 beats per minute. On examination, he has an audible S3 and a 3/5 pansystolic murmur heard loudest in the mitral area, his apex beat is displaced.

A recent echo showed a regurgitant mitral valve with evidence of left atrial and ventricular dilatation but a preserved ejection fraction.

His current medications include naproxen for osteoarthritis and over-the-counter vitamin D supplements.

From the following options, what would be the most appropriate next step?

○ Adopt a 'watch and wait' approach with serial yearly echocardiography

○ Commence bisoprolol

○ Commence sacubitril-valsartan

○ Refer for cardioversion

○ Refer for mitral valve replacement

Submit answer

Reference ranges

Score: **28%**

1	✗
2	✓
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Question 26 of 129 ✓

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A recent echo showed a regurgitant mitral valve with evidence of left atrial and ventricular dilatation but a preserved ejection fraction.

His current medications include naproxen for osteoarthritis and over-the-counter vitamin D supplements.

From the following options, what would be the most appropriate next step?

Adopt a 'watch and wait' approach with serial yearly echocardiography	17%
Commence bisoprolol	12%
Commence sacubitril-valsartan	3%
Refer for cardioversion	6%
Refer for mitral valve replacement	64%

New AF in mitral regurgitation -> refer for mitral valve replacement

Important for me Less important

Refer for mitral valve replacement is the correct answer. This patient has evidence of asymptomatic severe mitral regurgitation as indicated by narrow pulse pressure, audible S3 and evidence of left ventricular dilatation. Given he has now developed atrial fibrillation on a background of severe mitral regurgitation he should be referred for consideration of a valve repair or replacement.

Commence bisoprolol is incorrect. The patient in this scenario has a preserved ejection fraction as indicated by the echo findings. If there was evidence of reduced ejection fraction current NICE guidelines advises considering starting a beta-blocker and ACE inhibitor.

Commence sacubitril-valsartan is incorrect. This would be considered a second-line treatment for heart failure with reduced ejection fraction. In this case, the patient has a preserved ejection fraction.

Adopt a 'watch and wait' approach with serial yearly echocardiography is incorrect. This could be considered as per patient preference, but in the case of severe mitral regurgitation with the development of new atrial fibrillation patients should be offered a referral for mitral valve replacement.

Refer for cardioversion is incorrect. In this case, the patient has known mitral regurgitation. Mitral disease is associated with the development of atrial fibrillation due to the associated structural changes in the left atrium. There is a much higher chance of maintaining sinus rhythm with mitral valve replacement compared to cardioversion. Mitral valve replacement is therefore the preferred option.

👍 👎 Discuss (3) Improve

Next question ›

Mitral regurgitation ★

Also known as mitral insufficiency, mitral regurgitation (MR) occurs when blood leaks back through the mitral valve on systole. It is the second most common valve disease after aortic stenosis. The mitral valve is located between the left atrium and ventricle, and regurgitation leads to a less efficient heart as less blood is pumped through the body with each contraction. This said, MR is common in otherwise healthy patients to a trivial degree and does not need treatment.

As the degree of regurgitation becomes more severe, the body's oxygen demands may exceed what the heart can supply and as a result, the myocardium can thicken over time. While this may be benign initially, patients may find themselves increasingly fatigued as a thicker myocardium becomes less efficient, and eventually go into irreversible heart failure.

Risk factors

- Female sex
- Lower body mass
- Age
- Renal dysfunction
- Prior myocardial infarction
- Prior mitral stenosis or valve prolapse
- Collagen disorders e.g. Marfan's Syndrome and Ehlers-Danlos syndrome

Causes

- Following coronary artery disease or post-MI: if the papillary muscles or chordae tendinae are affected by a cardiac insult, mitral valve disease may ensue as a result of damage to its supporting structures.
- Mitral valve prolapse: Occurs when the leaflets of the mitral valve is deformed so the valve does not close properly and allows for backflow. Most patients with this have a trivial degree of mitral regurgitation.
- Infective endocarditis: When vegetations from the organisms colonising the heart grow on the mitral valve, it is prevented from closing properly. Patients with abnormal valves are more likely to develop endocarditis as opposed to their peers.
- Rheumatic fever: While this is uncommon in developed countries, rheumatic fever can cause inflammation of the valves and therefore result in mitral regurgitation.
- Congenital

Symptoms

- Most patients with MR are asymptomatic, and patients suffering from mild to moderate MR may stay largely asymptomatic indefinitely. Symptoms tend to be due to failure of the left ventricle, arrhythmias or pulmonary hypertension. This may present as fatigue, shortness of breath and oedema.

Signs

- The murmur heard on auscultation of the chest is typically a pansystolic murmur described as 'blowing'. It is heard best at the apex and radiating into the axilla. S1 may be quiet as a result of incomplete closure of the valve. Severe MR may cause a widely split S2

Investigations

- ECG may show a broad P wave, indicative of atrial enlargement
- Cardiomegaly may be seen on chest x-ray, with an enlarged left atrium and ventricle
- Echocardiography is crucial to diagnosis and to assess severity

Treatment options

- Medical management in acute cases involves nitrates, diuretics, positive inotropes and an intra-aortic balloon pump to increase cardiac output
- If patients are in heart failure, ACE inhibitors may be considered along with beta-blockers and spironolactone
- In acute, severe regurgitation, surgery is indicated
- The evidence for repair over replacement is strong in degenerative regurgitation, and is demonstrated through lower mortality and higher survival rates
- When this is not possible, valve replacement with either an artificial valve or a pig valve is considered

AI + Q 123

Next question >

Textbooks

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Extended textbook

Media

MITRAL REGURGITATION (INSUFFICIENCY)

Mitral regurgitation

Armando Hasudungan - YouTube 👍 5 👎 0

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 1 👎 0

Mitral regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1

Report broken media

Score: **30.8%**

1 ✗
2 ✓
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You are the on-call medical doctor called to review a patient in the Emergency Department. A 54 year old male patient with a past history asthma, ischaemic heart disease and transient ischaemic attack has presented with palpitations. His admission ECG shows a regular, narrow complex tachycardia. Vagal manoeuvres have been tried in the department with no success. You decide to give intravenous adenosine in an attempt to chemically cardiovert. An initial dose of 6mg is given into a proximal vein with a large flush. The patient soon loses consciousness and an 11 second ventricular standstill is noted on the rhythm strip before slow return of sinus rhythm.

Which of the patient's medications is most likely to be responsible for this?

- ○ Phyllocontin
- ○ Dipyridamole
- ○ Bisoprolol
- ○ Simvastatin
- ○ Montelukast

Submit answer

Reference ranges

Score: **30.8%**

1 ✗
2 ✓

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Which of the patient's medications is most likely to be responsible for this?

Phyllocontin	11%
Dipyridamole	46%
Bisoprolol	29%
Simvastatin	1%
Montelukast	13%

The effects of adenosine are enhanced by dipyridamole

Important for me Less important

Adenosine is a useful medication in the investigation and treatment of regular narrow complex tachycardias. With reference to the Resuscitation UK guidelines, it has a place as a second line measure to vagal manoeuvres when no 'adverse features' are present. Adenosine works by transiently blocking the AV node by effect on the A1 receptor, causing potassium efflux and hyperpolarisation.

Dont be side-tracked by the fact that this patient is asthmatic, yes this is a relative contraindication to the administration of adenosine, but that is not the question. Theophyllines have been known to antagonise adenosine necessitating higher dosages. Dipyridamole blocks the cellular uptake of adenosine, increasing concentration at receptors and potentiating its effect. Adenosine should therefore be used with caution, if at all, in patients taking dipyridamole, and certainly low starting doses should be considered.

Discuss (6) | Improve

Next question >

Adenosine

Adenosine is most commonly used to terminate supraventricular tachycardias. The effects of adenosine are enhanced by dipyridamole (antiplatelet agent) and blocked by theophyllines. It should be avoided in asthmatics due to possible bronchospasm.

Mechanism of action

- causes transient heart block in the AV node
- agonist of the A1 receptor in the atrioventricular node, which inhibits adenylyl cyclase thus reducing cAMP and causing hyperpolarization by increasing outward potassium flux
- adenosine has a very short half-life of about 8-10 seconds

Adenosine should ideally be infused via a large-calibre cannula due to it's short half-life,

Adverse effects

- chest pain
- bronchospasm
- transient flushing
- can enhance conduction down accessory pathways, resulting in increased ventricular rate (e.g. WPW syndrome)

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **29.6%**

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26	✓
27	✗

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Question 28 of 129

A 65-year-old man is referred to cardiology outpatient clinic with exertional shortness of breath. He becomes breathless after ascending a single flight of stairs. He has a background of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 63 beats per minute, blood pressure 120/77 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37ºC.

On examination, he is euvolemic. Chest auscultation is normal. There is no peripheral oedema. The pulse is regular.

From the listed options, what is the best medication choice to improve his symptoms?

○ Add ivabradine

○ Add nicorandil

○ Add sacubitril-valsartan

○ Replace ramipril with ivabradine

○ Replace ramipril with sacubitril-valsartan

Submit answer

Reference ranges

Score: **29.6%**

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Question 28 of 129 ✓

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His observations are heart rate 63 beats per minute, blood pressure 120/77 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37ºC.

On examination, he is euvolemic. Chest auscultation is normal. There is no peripheral oedema. The pulse is regular.

From the listed options, what is the best medication choice to improve his symptoms?

Add ivabradine	6%
Add nicorandil	4%
Add sacubitril-valsartan	12%
Replace ramipril with ivabradine	1%
Replace ramipril with sacubitril-valsartan	77%

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & beta-blockers

Important for me Less important

Replace ramipril with sacubitril-valsartan is the correct answer. This man has NYHA class II/III heart failure not controlled with an ace inhibitor, beta blocked and mineralocorticoid receptor antagonist. Given his ejection fraction is < 35%, replacing ramipril with sacubitril-valsartan is a suitable option to improve the clinical situation.

Add ivabradine is incorrect. This would be an option if his heart rate was > 75 beats per minute.

Add sacubitril-valsartan is incorrect. This should replace an ace inhibitor rather than be added to this treatment.

Replace ramipril with ivabradine is incorrect. This is not a substitution recommended by guidance and the patient's heart rate is too low for the addition of ivabradine.

Add nicorandil is incorrect. This medication is used in the treatment of stable angina, rather than heart failure.

👍 👎 Discuss (4) Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**
- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

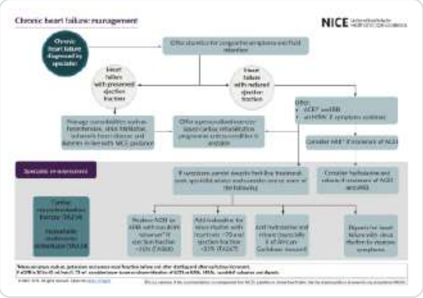
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients

- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years



123

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NICE 14 13

2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

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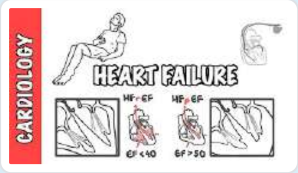
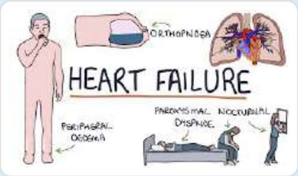

CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
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PAROXYSMAL NOCTURNAL DYSPNOEA
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Heart failure

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Score: **32.1%**

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2 ✓

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28	✓

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Question 29 of 129

A 72-year-old man is reviewed in the cardiology outpatient department with worsening shortness of breath, which now occurs at rest. He has a past medical history of heart failure with reduced ejection fraction and ischaemic heart disease. His medications include bisoprolol, ramipril, and spironolactone.

On examination, he is clinically euvolemic.

An ECG demonstrates a QRS of 140ms and a left bundle branch block.

What intervention is most likely to improve the patient's symptoms?

- ○ Amiodarone
- ○ Biventricular pacemaker
- ○ Dual chamber pacemaker
- ○ Implantable cardiac defibrillator
- ○ Single chamber atrial pacemaker

Submit answer

Reference ranges

Score: **32.1%**

1 ✗
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3 ✗

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On examination, he is clinically euvolemic.

An ECG demonstrates a QRS of 140ms and a left bundle branch block.

What intervention is most likely to improve the patient's symptoms?

Amiodarone	2%
Biventricular pacemaker	50%
Dual chamber pacemaker	27%
Implantable cardiac defibrillator	20%
Single chamber atrial pacemaker	1%

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

Important for me Less important

Biventricular pacemaker is correct. Cardiac synchronization therapy (CRT) with a biventricular pacemaker has been shown to improve heart failure symptoms in those patients with a QRS duration > 130ms and LBBB morphology, inadequately controlled on optimal medical therapy. He has NYHA class IV heart failure as he has symptoms at rest. Typically, leads of this pacemaker are placed in both ventricles and often one in the right atrium in addition. They are designed to help the heart contract more efficiently.

Amiodarone is incorrect. This is typically used to control abnormal tachyarrhythmias. It has not been shown to improve symptoms in patients with chronic heart failure.

Dual chamber pacemaker is incorrect. This type of pacemaker paces both the right atrium and right ventricle. It is indicated in conditions such as high-grade atrioventricular block rather than advanced heart failure as in this patient.

Implantable cardiac defibrillator is incorrect. This is typically indicated in patients with serious ventricular arrhythmias at risk of sudden death. It can be used for some patients with NYHA class I-III heart failure and a prolonged QRS 120-149ms but without LBBB. However, if there is an LBBB morphology as in this case then a CRT is preferred to improve the symptoms of heart failure.

Single chamber atrial pacemaker is incorrect. This is a pacemaker that usually paces just the right atrium. This type of pacemaker has largely fallen out of favour as they require intact atrioventricular conduction and do not safeguard against this failure in the future. Typically, this type of pacing would only be used for severe symptomatic sinus bradycardia or sick sinus syndrome. It would not result in the required synchronization of ventricular contraction which would result in improved symptoms of heart failure.

👍 👎 Discuss (2) Improve

Next question >

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy
- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training
- improves symptoms but not hospitalisation/mortality

123

Next question >

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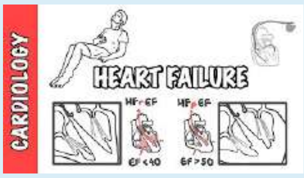
NICE 👍 10 👎 1

Cardiac resynchronisation therapy for the treatment of heart failure

Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
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Heart failure with reduced and preserved ejection fraction

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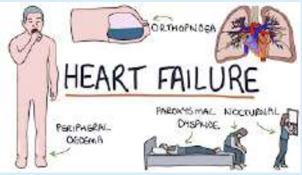
ORTHOPNOEA
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PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

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Score: **31%**

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Question 30 of 129

A 56-year-old gentleman has presented with central chest tightness with a strangling feeling in his jaw. This started four hours ago and he has become increasingly breathless. He feels clammy and has vomited twice. He felt well prior to today and only has a history of refractory hypertension.

On examination, he is alert with a Glasgow Coma Scale of 14/15. He is clutching his chest with one hand. Both heart sounds are present with a loud pan systolic murmur and there is air entry heard throughout. His JVP is raised at 4cm and there are cannon V waves present. There is no ankle swelling or oedema. His liver is palpable and pulsatile. His blood pressure is 190/110 mmHg, heart rate 90/min and he is saturating at 92% in

Hb	130 g/l	Na^+	139 mmol/l
Platelets	$356 * 10^9/l$	K^+	4.6 mmol/l
WBC	$13.4 * 10^9/l$	Urea	5.4mmol/l
Neuts	$9.4 * 10^9/l$	Creatinine	76 µmol/l
Lymphs	$2.3 * 10^9/l$	CRP	14 mg/l
Troponin	1640 ng/ml (normal range <20)		

ECG	ST depression in leads V1-V3, sinus rhythm
Chest X-ray	enlarged cardiac shadow

What is the likely diagnosis?

- ○ Infective endocarditis ×
- ○ Right ventricle myocardial infarction ×
- ○ Anteroseptal myocardial infarction ×
- ○ Dilated cardiomyopathy ×

○ Aortic dissection ×

Submit answer

Reference ranges ⌵

Score: **31%**

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Neuts	9.4 * 10^9/l	Creatinine	76 µmol/l
Lymphs	2.3 * 10^9/l	CRP	14 mg/l
Troponin	1640 ng/ml (normal range <20)		

ECG	ST depression in leads V1-V3, sinus rhythm
Chest X-ray	enlarged cardiac shadow

What is the likely diagnosis?

Infective endocarditis	2%
Right ventricle myocardial infarction	43%
Anteroseptal myocardial infarction	21%
Dilated cardiomyopathy	5%

Aortic dissection 29%

Right ventricle infarction -> tricuspid regurgitation

Important for me Less important

This gentleman presents with acute onset chest pain and has been well prior to this. Infective endocarditis would present more insidiously and is typically painless. Cardiomyopathy does not present with chest pain. Aortic dissection usually has a tearing pain that radiates to the back. The ECG changes represent reciprocal changes seen in a posterior myocardial infarction. The cannon V waves are pathognomonic for a right sided heart failure which occurs during infarction of the right ventricle. Furthermore, the pansystolic murmur would reflect tricuspid regurgitation. Both these features occur in right ventricle infarcts.

Discuss (13) Improve

Next question >

Tricuspid regurgitation ★

Signs

- pan-systolic murmur
- prominent/giant V waves in JVP
- pulsatile hepatomegaly
- left parasternal heave

Causes

- right ventricular infarction
- pulmonary hypertension e.g. COPD
- rheumatic heart disease
- infective endocarditis (especially intravenous drug users)
- Ebstein's anomaly
- carcinoid syndrome

AI + Q 123

Next question ›

B *I*

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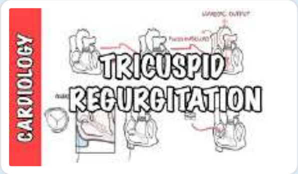
CARDIOLOGY
TRICUSPID
REGURGITATION

TyszyqQhBfc

Armando Hasudungan - YouTube 👍 1 👎 1

Tricuspid valve disease

Osmosis - YouTube 👍 1 👎 1

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Score: **30%**

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Question 31 of 129

You review a patient in cardiology outpatient clinic. He has a left ventricular ejection fraction of 15% on maximal medical therapy. On reviewing his ECG you note a QRS duration of 135ms without evidence of left bundle branch block. Upon taking a history you note the patient is breathless at rest and struggles to walk around his bungalow.

Which implantable device would be appropriate to consider?

- ○ Cardiac resynchronisation therapy without defibrillator
- ○ Cardiac resynchronisation therapy with defribrillator
- ○ VVI pacemaker
- ○ No implantable device recommended
- ○ Implantable cardioverter defibrillator alone

Submit answer

Reference ranges

Score: **30%**

1 ✗
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7 ✓

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31	-

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Which implantable device would be appropriate to consider?

- Cardiac resynchronisation therapy without defibrillator 37%
- Cardiac resynchronisation therapy with defribrillator 46%
- VVI pacemaker 5%
- No implantable device recommended 5%
- Implantable cardioverter defibrillator alone 7%

The patient has New York Heart Association Class IV symptoms as he is limited by breathlessness at rest and on minimal exertion. The NICE guidance on implantable devices and resynchronisation therapy would suggest cardiac resynchronisation therapy (without defibrillator) in the above case as QRS is between 120 and 149ms without evidence of left bundle branch block.

VVI pacing is useful in atrial fibrillation with slow ventricular response.

NICE TA314: Implantable cardioverter defibrillators and cardiac resynchronisation therapy for arrhythmias and heart failure.

https://www.nice.org.uk/guidance/TA314/chapter/1-Guidance

Discuss (22) | Improve

Next question

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy
- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training
- improves symptoms but not hospitalisation/mortality

AI + Q 123

Next question >

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Cardiac resynchronisation therapy for the treatment of heart failure

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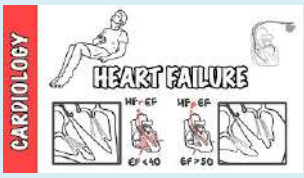
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HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

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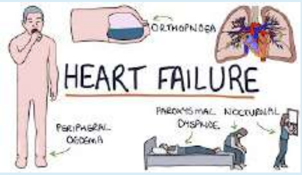
ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

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Score: **29%**

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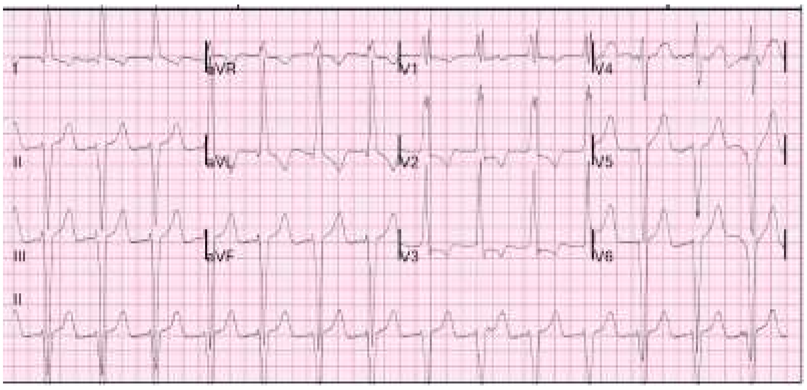
A 70-year-old presents to the outpatients' department having been referred by his GP with on-off palpitations for several months. The patient has a myocardial infarct over 20 years ago which was successfully managed via thrombolysis but otherwise, he has no significant past medical history.

The patient reports he has spontaneous episodes of palpitations approximately once every 2 weeks, and occasional feeling faint. He fainted once when walking quickly for the bus and so now takes it slowly when exerting himself and has no further similar episodes. He is otherwise very well and denies any episodes of chest pain etc.

He is on secondary preventive medication including:

- Aspirin low dose
- Bisoprolol 10mg twice daily
- Atorvastatin 40mgs once daily
- Ramipril 10mg once daily

His examination and observation are normal and an ECG performed is shown below.



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What is the most appropriate management plan for the patient?

○ Admit for urgent ablation workup ×

○ Admit for urgent pacemaker workup ×

○ No additional management is required ×

○ Reduce the dose of his beta-blocker ×

○ Arrange for urgent 24-hour Holter monitor ×

Submit answer

Reference ranges ∨

Score: **29%**

1	✗
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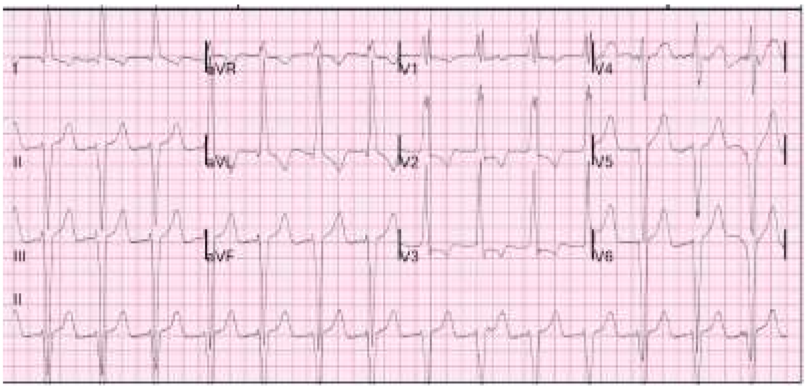

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What is the most appropriate management plan for the patient?

Admit for urgent ablation workup 18%

Admit for urgent pacemaker workup 38%

No additional management is required	8%
Reduce the dose of his beta-blocker	9%
Arrange for urgent 24-hour Holter monitor	27%

The patient's ECG show trifascicular block with a right bundle branch block (RBBB), left anterior hemiblock and 1st-degree heart block. This with the history of pre-syncope and syncope is concerning and is an indication for admission and pacemaker workup as the patient is at risk of further significant arrhythmias or cardiac blocks.

Trifascicular block is a ventricular escape rhythm that usually arises from the left anterior or posterior fascicle regions following ischaemic heart disease or structural heart disease. As such ablation is not a viable treatment option. Symptomatic trifascicular block is an indication for implantable cardiac pacemaker or permanent pacemaker work up.

The patient has concerning features of symptomatic trifascicular block with pre-syncope and syncope on exertion which warrants urgent assessment for a pacemaker.

The patient's trifascicular block is very unlikely to be associated with his beta-blocker medication and therefore reducing the dose will not remove the risk of further significant arrhythmias or cardiac blocks.

Arranging for an urgent 24-hour Holter monitor is unnecessary as the patient's ECG is unlikely to change and the current risk of arrhythmias or heart block requires urgent intervention.

Discuss (9) Improve

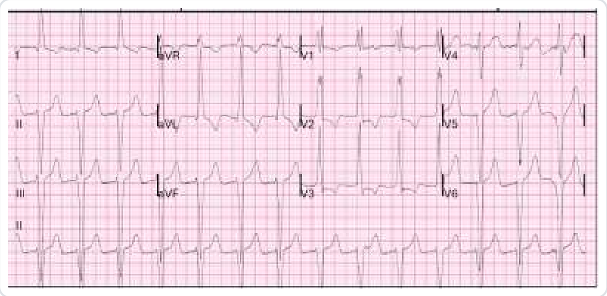
Next question >

ECG: bi/tri-fascicular block ★

Bifascicular block
- the combination of RBBB with left anterior or posterior hemiblock
- e.g. RBBB with left axis deviation

Trifascicular block

- features of bifascicular block as above + 1st-degree heart block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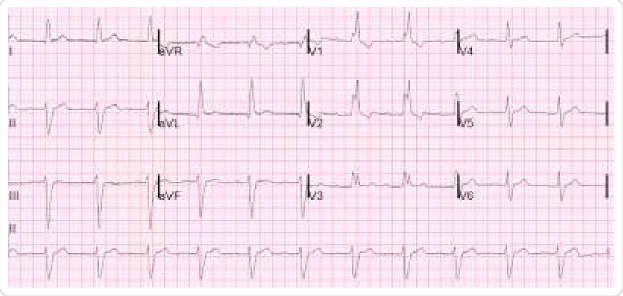
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Life in the fast lane 7 4

[Bifascicular block]

Life in the fast lane 👍 7 👎 0

Trifascicular block

Suggest link Report broken link

Score: **28.1%**

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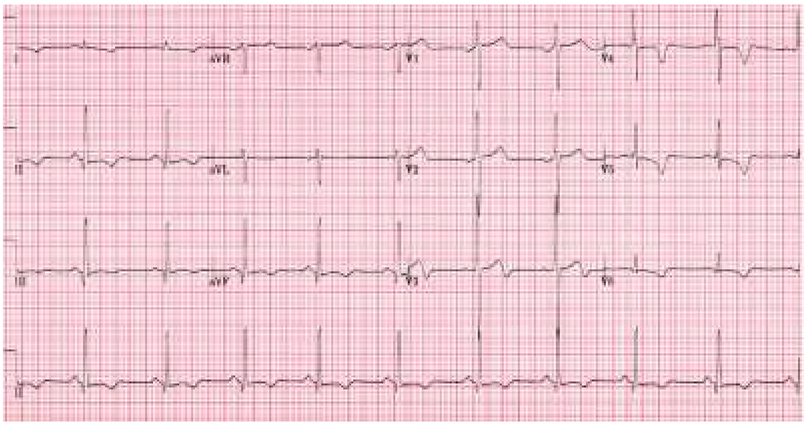
A 34-year-old is brought into the emergency department following a syncope episode whilst playing tennis. The patient reports he is now feeling well with no symptoms. On further questioning, he only plays tennis occasionally and does not exercise regularly. He reports experienced shortness of breath and chest discomfort when playing and has had a few episodes of feeling light-headed but has never fainted before. He is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



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What is the patient's most likely diagnosis?

○ Brugada syndrome

○ Dilated cardiomyopathy

○ Hypertrophic obstructive cardiomyopathy

○ Arrhythmogenic right ventricular dysplasia (ARVD)

○ Wellen's syndrome

Submit answer

31	✗
32	✗
33	-

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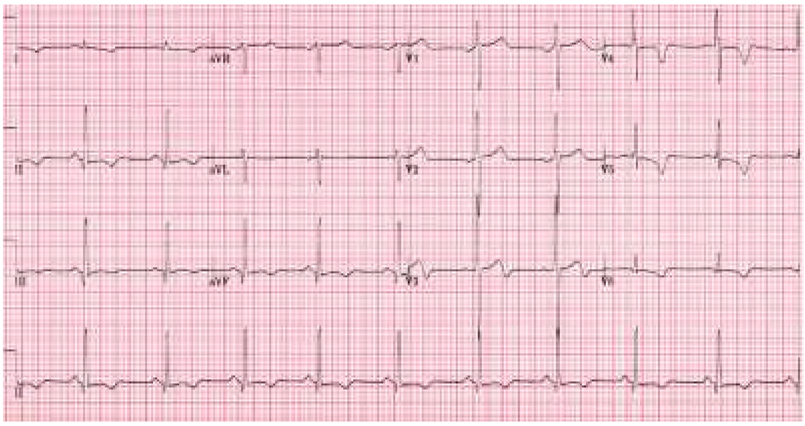
A 34-year-old is brought into the emergency department following a syncope episode whilst playing tennis. The patient reports he is now feeling well with no symptoms. On further questioning, he only plays tennis occasionally and does not exercise regularly. He reports experienced shortness of breath and chest discomfort when playing and has had a few episodes of feeling light-headed but has never fainted before. He is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



© Image used on license from Dr Smith, University of Minnesota

What is the patient's most likely diagnosis?

Brugada syndrome	21%
Dilated cardiomyopathy	2%
Hypertrophic obstructive cardiomyopathy	45%
Arrhythmogenic right ventricular dysplasia (ARVD)	18%
Wellen's syndrome	14%

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect that causes a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncopal episodes, commonly following or during exertion. ECG findings are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. On this patient's ECG widespread T wave inversion can be seen, especially in lead I & II as well as non-specific ST-segment changes in V2 & 3. The diagnosis can be confirmed via cardiac echo and management is dependant on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome, each with variations seen on ECG. However, the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as the Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Arrhythmogenic right ventricular dysplasia (ARVD), also known as arrhythmogenic right ventricular cardiomyopathy (ARVC), is a result of a genetic defect affecting the desmosomes of the myocardium. ARVD results in non-ischemic cardiomyopathy, mainly affecting the right ventricle. The condition leads to hypokinetic areas of the ventricle wall and myocardium fibrofatty replacement with which results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

👍 👎 Discuss (6) Improve

Next question >

Hypertrophic obstructive cardiomyopathy: features ★

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500. HOCM is important as it is the most common cause of sudden cardiac death in the young.

Pathophysiology

- the most common defects involve a mutation in the gene encoding β-myosin heavy chain protein or myosin-binding protein C
- results in predominantly diastolic dysfunction
  - left ventricle hypertrophy → decreased compliance → decreased cardiac output
- characterized by myofibrillar hypertrophy with chaotic and disorganized fashion myocytes ('disarray') and fibrosis on biopsy

Features

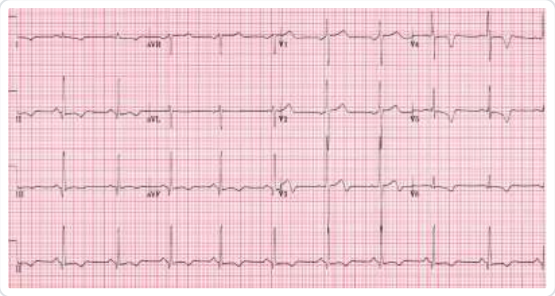
- often asymptomatic
- exertional dyspnoea
- angina
- syncope
  - typically following exercise
  - due to subaortic hypertrophy of the ventricular septum, resulting in functional aortic stenosis
- sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure
- jerky pulse, large 'a' waves, double apex beat
- systolic murmurs
  - ejection systolic murmur: due to left ventricular outflow tract obstruction. Increases with Valsalva manoeuvre and decreases on squatting

    - pansystolic murmur: due to systolic anterior motion of the mitral valve → mitral regurgitation

Associations
- Friedreich's ataxia
- Wolff-Parkinson White

Echo findings - mnemonic - MR SAM ASH
- mitral regurgitation (MR)
- systolic anterior motion (SAM) of the anterior mitral valve leaflet
- asymmetric hypertrophy (ASH)

ECG
- left ventricular hypertrophy
- non-specific ST segment and T-wave abnormalities, progressive T wave inversion may be seen
- deep Q waves
- atrial fibrillation may occasionally be seen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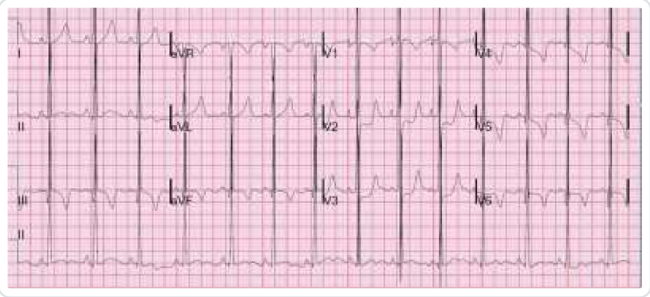
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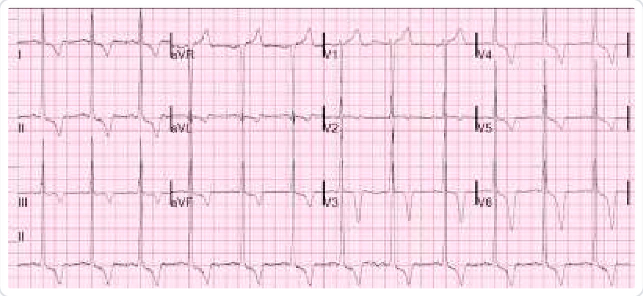
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II

123

Next question >

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology (ESC) 2014 ESC HCOM Guidelines	👍 9	👎 8
RCP Investigation and treatment of hypertrophic cardiomyopathy	👍 16	👎 4

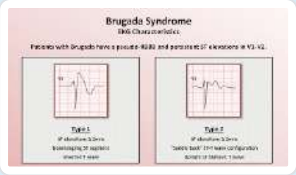
Suggest link

Report broken link

Media

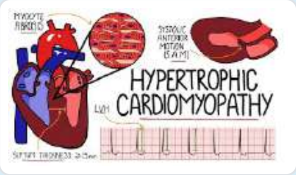
Hypertrophic cardiomyopathy

Osmosis - YouTube	👍 0	👎 0


Brugada Syndrome

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 6 👎 1


HYPERTROPHIC
CARDIOMYOPATHY

Hypertrophic Cardiomyopathy Made Easy

Rhesus Medicine - YouTube 👍 1 👎 1

Hypertrophic Cardiomyopathy heart sounds - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 1 👎 1

CARDIOLOGY
HYPERTROPHIC
CARDIOMYOPATHY

Hypertrophic obstructive cardiomyopathy

Osmosis - YouTube 👍 0 👎 4

Report broken media

Score: **27.3%**

1 ✗
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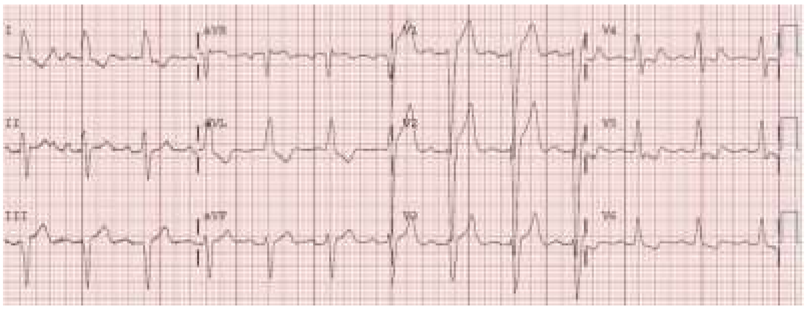
Question 34 of 129

A 76-year-old man is brought to the Emergency Department by the paramedics after experiencing an episode of chest pain. This came whilst he was walking his dog and lasted around 20 minutes. He is now pain free. The paramedics report that his blood pressure is 126/88mmHg, pulse 75/min and oxygen saturations 98% on room air. On examination you note a systolic murmur. An ECG is requested:



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What does the ECG show?

○ Hypertrophic obstructive cardiomyopathy

○ Anterior myocardial infarction

○ Left bundle branch block

○ Brugada syndrome

○ Arrhythmogenic right ventricular cardiomyopathy

Submit answer

Reference ranges

Score: **27.3%**

1	✗
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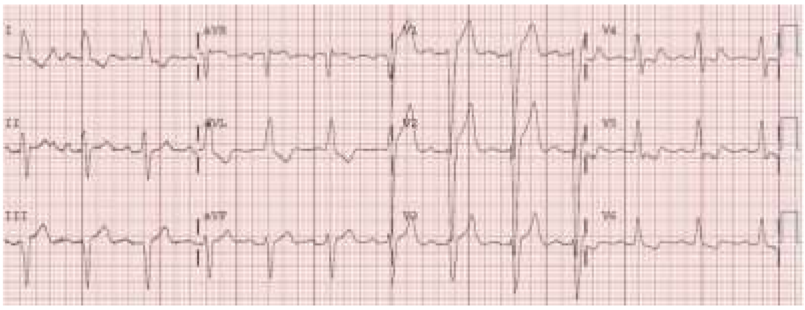
Question 34 of 129 ×

A 76-year-old man is brought to the Emergency Department by the paramedics after experiencing an episode of chest pain. This came whilst he was walking his dog and lasted around 20 minutes. He is now pain free. The paramedics report that his blood pressure is 126/88mmHg, pulse 75/min and oxygen saturations 98% on room air. On examination you note a systolic murmur. An ECG is requested:



© Image used on license from Dr Smith, University of Minnesota

What does the ECG show?

Hypertrophic obstructive cardiomyopathy	11%
Anterior myocardial infarction	33%
Left bundle branch block	44%
Brugada syndrome	8%
Arrhythmogenic right ventricular cardiomyopathy	5%

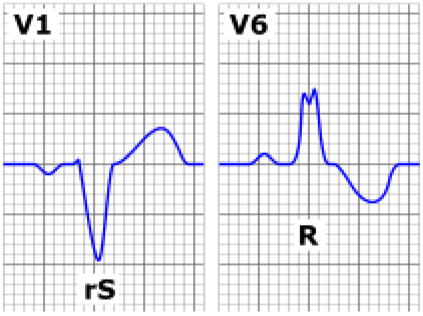
The ECG shows typical changes of left bundle branch block (LBBB). The systolic murmur may be explained by aortic stenosis, a known association of LBBB. Cardiac ischaemia should of course be considered in patients with LBBB, particularly if it is new. A troponin level will help clarify matters but patients with ongoing chest pain and LBBB should be transferred to the cath lab.

Discuss (8) | Improve

Next question >

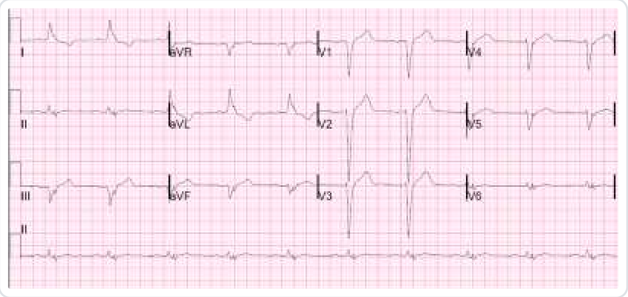
ECG: left bundle branch block

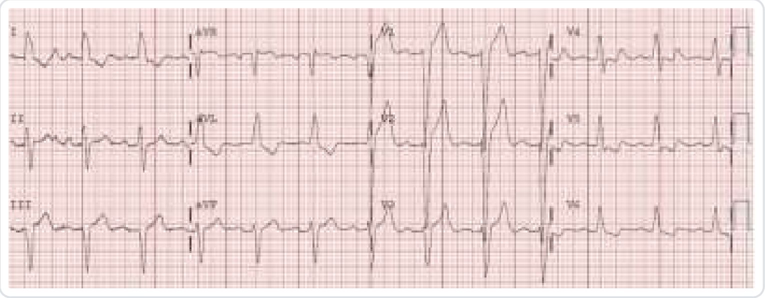
The diagram below shows the typical features of left bundle branch block (LBBB):



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6





New LBBB is always pathological. Causes of LBBB include:

- myocardial infarction
    - diagnosing a myocardial infarction for patients with *existing* LBBB is difficult
    - rhe Sgarbossa criteria can help with this - please see the link for more details

- hypertension
- aortic stenosis
- cardiomyopathy
- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

123

Next question >

Textbooks

High-yield textbook

Extended textbook

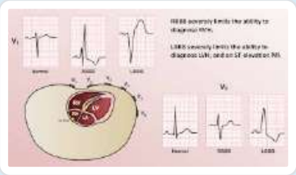
Links

Life in the Fast Lane 👍 33 👎 16

Sgarbossa criteria

Suggest link Report broken link

Media



Intro to EKG Interpretation - Bundle Branch Blocks

Strong Medicine - YouTube 👍 6 👎 2

Bundle branch block

Osmosis - YouTube 👍 0 👎 3

Report broken media

Score: **26.5%**

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33	✗
34	✗

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Question 35 of 129

A 66-year-old gentleman requests to see his general practitioner, concerned regarding one of the medications that he was started on following a myocardial infarction. He tells you that the cardiology team has started him on atorvastatin, which he does not wish to take unless there is no other alternative, as he has had relatives that had bad experience with simvastatin, including muscle pain and disturbed sleep.

He has no family history of hypercholesterolaemia at a young age, but a number of his relatives are on statins following cardiac events.

What options exist with regards to his secondary prevention?

- ○ Ezetimibe 10 mg OD
- ○ Continue with atorvastatin at the current dose
- ○ Fenofibrate 160 mg OD
- ○ Lomitapide 5 mg OD
- ○ Niacin (as per product literature)

Submit answer

Reference ranges

Score: **26.5%**

1 ✗
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Question 35 of 129 ✗

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He has no family history of hypercholesterolaemia at a young age, but a number of his relatives are on statins following cardiac events.

What options exist with regards to his secondary prevention?

Ezetimibe 10 mg OD	44%
Continue with atorvastatin at the current dose	44%
Fenofibrate 160 mg OD	9%
Lomitapide 5 mg OD	2%
Niacin (as per product literature)	2%

Statins are the only lipid-regulating drugs that are used in secondary prevention of cardiovascular disease (with the exception of ezetimibe in cases of primary hypercholesterolaemia)

Important for me Less important

With regards to secondary prevention the only NICE approved medication at the moment is statins. None of the other drugs mentioned above are licensed for secondary prevention with the exception of ezetimibe in primary hypercholesterolaemia. There is no indication, in a 66 year old gentleman that he is suffering from primary hypercholesterolaemia. As no dose is given in the question, one should assume that continuing at the current dose provided that the patient understands that no alternative exists is the right answer. Indeed if a dose was given, reducing the dose or switching to an even more hydrophilic agent (such as pravastatin) would be an appropriate option.

👍 👎 Discuss (10) Improve

Next question ›

Statins ★

Statins inhibit the action of HMG-CoA reductase, the rate-limiting enzyme in hepatic cholesterol synthesis.

Adverse effects

- myopathy: includes myalgia, myositis, rhabdomyolysis and asymptomatic raised creatine kinase. Risks factors for myopathy include advanced age, female sex, low body mass index and presence of multisystem disease such as diabetes mellitus. Myopathy is more common in lipophilic statins (simvastatin, atorvastatin) than relatively hydrophilic statins (rosuvastatin, pravastatin, fluvastatin)
- liver impairment: the 2014 NICE guidelines recommend checking LFTs at baseline, 3 months and 12 months. Treatment should be discontinued if serum transaminase concentrations rise to and persist at 3 times the upper limit of the reference range
- there is some evidence that statins may increase the risk of intracerebral haemorrhage in patients who've previously had a stroke. This effect is not seen in primary prevention. For this reason the Royal College of Physicians recommend avoiding statins in patients with a history of intracerebral haemorrhage

Contraindications

- macrolides (e.g. erythromycin, clarithromycin) are an important interaction. Statins should be stopped until patients complete the course
- pregnancy

Who should receive a statin?

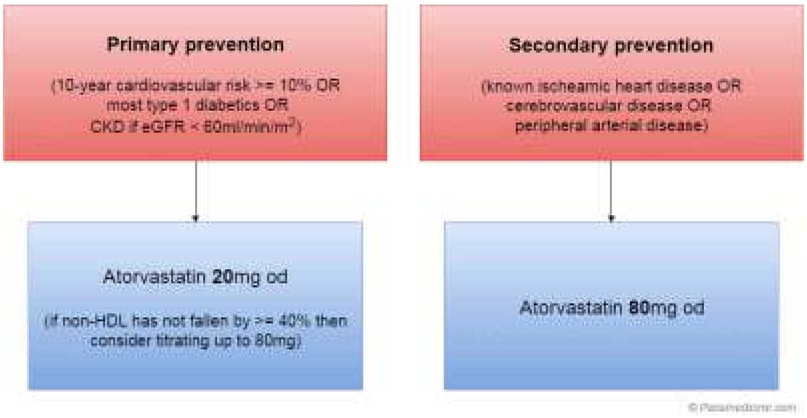
- all people with established cardiovascular disease (stroke, TIA, ischaemic heart disease, peripheral arterial disease)

- following the 2014 update, NICE recommend anyone with a 10-year cardiovascular risk >= 10%
- patients with type 2 diabetes mellitus should now be assessed using QRISK2 like other patients are, to determine whether they should be started on statins
- patients with type 1 diabetes mellitus who were diagnosed more than 10 years ago OR are aged over 40 OR have established nephropathy

Statins should be taken at night as this is when the majority of cholesterol synthesis takes place. This is especially true for simvastatin which has a shorter half-life than other statins.

NICE currently recommends the following for the prevention of cardiovascular disease::

- atorvastatin 20mg for primary prevention
  - increase the dose if non-HDL has not reduced for >= 40%
- atorvastatin 80mg for secondary prevention


Primary prevention
(10-year cardiovascular risk >= 10% OR most type 1 diabetics OR CKD if eGFR < 60ml/min/m²)
Secondary prevention
(known ischeamic heart disease OR cerebrovascular disease OR peripheral arterial disease)
Atorvastatin 20mg od
(if non-HDL has not fallen by >= 40% then consider titrating up to 80mg)
Atorvastatin 80mg od

Graphic showing choice of statin.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

RCP 👍 15 👎 21

2016 Stroke guidelines

NICE 👍 20 👎 16

2014 Lipid modification guidelines

Clinical Knowledge Summaries 👍 18 👎 14

Lipids management

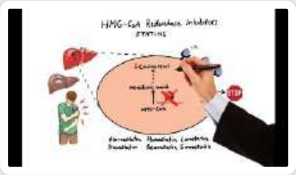
NICE 👍 18 👎 12

2022 Type 2 diabetes guidelines

Suggest link

Report broken link

Media



Drugs for hyperlipidaemia

Speed Pharmacology - YouTube 👍 3 👎 0

Report broken media

Score: **25.7%**

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35 ✗

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Question 36 of 129

A 50-year-old patient with no prior medical history presents to the Emergency Department with crushing central chest pain. On examination you find:

- Respiratory rate - 22/min
- Oxygen saturations 95% on room air
- Chest clear
- Pulse 118bpm
- Blood pressure 92/61mmHg
- JVP elevated 4cm above sternal angle

An ECG done in the department shows 3mm ST segment elevation in leads II, III and aVF. The patient undergoes a primary PCI to his right coronary artery with a good angiographic result. An echocardiogram is performed the following day. What would you expect to see?

○ Regional wall motion abnormality in the inferior wall of the LV

○ Regional wall motion abnormality in the inferior wall of the LV with a dilated and impaired RV

○ Normal biventricular function

○ Constrictive pericarditis

○ Regional wall motion abnormality in the inferior wall of the LV with mitral regurgitation

Submit answer

Reference ranges

Score: **25.7%**

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36	-

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- Respiratory rate - 22/min
- Oxygen saturations 95% on room air
- Chest clear
- Pulse 118bpm
- Blood pressure 92/61mmHg
- JVP elevated 4cm above sternal angle

An ECG done in the department shows 3mm ST segment elevation in leads II, III and aVF. The patient undergoes a primary PCI to his right coronary artery with a good angiographic result. An echocardiogram is performed the following day. What would you expect to see?

Regional wall motion abnormality in the inferior wall of the LV	32%
Regional wall motion abnormality in the inferior wall of the LV with a dilated and impaired RV	43%
Normal biventricular function	12%
Constrictive pericarditis	2%
Regional wall motion abnormality in the inferior wall of the LV with mitral regurgitation	10%

This patient has had an inferior STEMI, as evidenced by the fact that he has ST elevation is leads II, III, aVf. Given this presentation, you would expect an echocardiogram to show a regional wall motion abnormality (RWMA) in the inferior wall of the LV. However, there are some additional key points in his examination findings that are necessary to appreciate in order to correctly answer this question. Firstly, he has significant hypotension which is not usual in the setting of an uncomplicated inferior STEMI. In addition, the patient has an elevated

JVP which is indicative of elevated RV filling pressures and therefore RV failure. The fact that he has a clear chest indicates that he has relatively normal LV filling pressures, thus ruling out RV failure secondary to LV failure. Putting these findings together, the diagnosis is an inferior STEMI with right ventricular infarction. Therefore you would expect an echo to demonstrate an inferior LV wall RWMA with a dilated and impaired RV.

👍 👎 Discuss (10) Improve

Next question >

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and	Usually left circumflex, also right coronary

	ECG changes	Coronary artery
	Q waves in posterior leads (V7-9)	

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

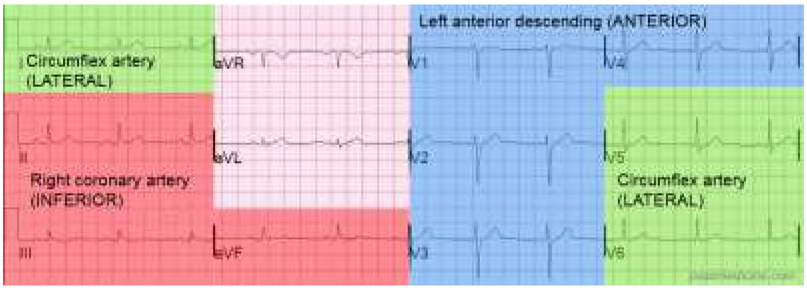
Circumflex artery (LATERAL)
I
aVR
Left anterior descending (ANTERIOR)
V1
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal	7	11

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 2 👎 6

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **25%**

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A 74-year-old man was admitted with chest pain. He had a past history of type 2 diabetes mellitus and hypertension. He underwent coronary angiography and a stent was inserted into his right coronary artery. Following the procedure he was noted to be oliguric.

On examination his blood pressure was 148/80 mmHg and he was euvolaemic. Mottled skin changes were noted over his legs.

Na^+	138 mmol/L	(135 - 145)
K^+	5.6 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	17.6 mmol/L	(2.0 - 7.0)
Creatinine	228 µmol/L	(55 - 120)
Creatine kinase	560 U/L	(24-170)

Hb	142 g/L	Male: (135-180) Female: (115 - 160)
Platelets	380 * 10^9/L	(150 - 400)
WBC	13.6 * 10^9/L	(4.0 - 11.0)
Neuts	9.1 * 10^9/L	(2.0 - 7.0)
Lymphs	2.5 * 10^9/L	(1.0 - 3.5)
Eosin	1.2 * 10^9/L	(0.0 - 0.4)

What is the most likely diagnosis?

○ Contrast-induced nephropathy ×

○ Acute interstitial nephritis ×

○ Cholesterol emboli ×

○ ANCA associated vasculitis ×

○ Rhabdomyolysis ×

Submit answer

Reference ranges ⌄

Score: **25%**

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Creatine kinase	560 U/L	(24-170)

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Platelets	380 * 10^9/L	(150 - 400)
WBC	13.6 * 10^9/L	(4.0 - 11.0)
Neuts	9.1 * 10^9/L	(2.0 - 7.0)
Lymphs	2.5 * 10^9/L	(1.0 - 3.5)
Eosin	1.2 * 10^9/L	(0.0 - 0.4)

What is the most likely diagnosis?

Contrast-induced nephropathy	9%
Acute interstitial nephritis	4%
Cholesterol emboli	85%
ANCA associated vasculitis	1%

Rhabdomyolysis 1%

AKI following angiography can be caused by contrast-induced nephropathy or cholesterol emboli

Important for me Less important

Although the commonest cause of acute kidney injury following angiography is contrast-induced nephropathy, the raised eosinophil count and skin rash (livedo reticularis) is highly suggestive of cholesterol emboli.

Acute interstitial nephritis can cause eosinophilia but is not associated with contrast.

ANCA associated vasculitis can cause a rash and eosinophilia (e.g. Churg Strauss) but there are no other features such as anaemia or systemic upset to fit with this.

The elevated creatine kinase suggests muscle injury, which would fit with an embolic phenomenon but the CK is not high enough to diagnose rhabdomyolysis (usually >5000).

Discuss (4) Improve

Next question

Cholesterol embolisation

Overview

- cholesterol emboli may break off causing renal disease
- the majority of cases are secondary to vascular surgery or angiography. Other causes include severe atherosclerosis, particularly in large arteries such as the aorta

Features

- eosinophilia

- purpura
- renal failure
- livedo reticularis

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **24.3%**

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A 59-year-old man presents with stridor which came on gradually over the course of the day. On examination you note swelling of the tongue and the mucous membranes of the oropharynx. He has a past medical history of rheumatoid arthritis, type 2 diabetes mellitus and heart failure.

He was recently started on a new medication. What medicine would be the most likely cause of this presentation?

- ○ Nimodipine
- ○ Losartan
- ○ Lisinopril
- ○ Prednisolone
- ○ Sitagliptin

Submit answer

Reference ranges

Score: **24.3%**

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Question 38 of 129 ✗

A 59-year-old man presents with stridor which came on gradually over the course of the day. On examination you note swelling of the tongue and the mucous membranes of the oropharynx. He has a past medical history of rheumatoid arthritis, type 2 diabetes mellitus and heart failure.

He was recently started on a new medication. What medicine would be the most likely cause of this presentation?

Nimodipine	12%
Losartan	4%
Lisinopril	82%
Prednisolone	1%
Sitagliptin	1%

Drug-induced angioedema without urticaria is most frequently associated with angiotensin converting enzyme inhibitors or, less frequently, angiotensin II receptor blockers

Important for me Less important

Angioedema is a well recognised side effect of angiotensin-converting enzyme (ACE) inhibitor therapy. Angioedema can also be seen with angiotensin receptor blocker (ARB) therapy but much less frequently than is the case with ACE inhibitors. Therefore lisinopril is a better answer than losartan.

ACE inhibitors block the enzyme ACE resulting in increased levels of bradykinin. Bradykinin then accumulates potentially resulting in angioedema.

All the other options can cause peripheral oedema, however not specifically angioedema as described.

👍 | 👎 | Discuss (1) | Improve

Next question >

ACE inhibitors ★

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in the secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibits the conversion angiotensin I to angiotensin II
  - → decrease in angiotensin II levels → to vasodilation and reduced blood pressure
  - → decrease in angiotensin II levels → reduced stimulation for aldosterone release → decrease in sodium and water retention by the kidneys
- renoprotective mechanism
  - angiotensin II constricts the efferent glomerular arterioles
  - ACE inhibitors therefore lead to dilation of the efferent arterioles → reduced glomerular capillary pressure → decreased mechanical stress on the delicate filtration barriers of the glomeruli
  - this is particularly important in diabetic nephropathy
- ACE inhibitors are activated by phase 1 metabolism in the liver

Side-effects:

- cough
  - occurs in around 15% of patients and may occur up to a year after starting treatment
  - thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications

- pregnancy and breastfeeding - avoid
- renovascular disease - may result in renal impairment
- aortic stenosis - may result in hypotension
- hereditary of idiopathic angioedema
- specialist advice should be sought before starting ACE inhibitors in patients with a potassium >= 5.0 mmol/L

Interactions

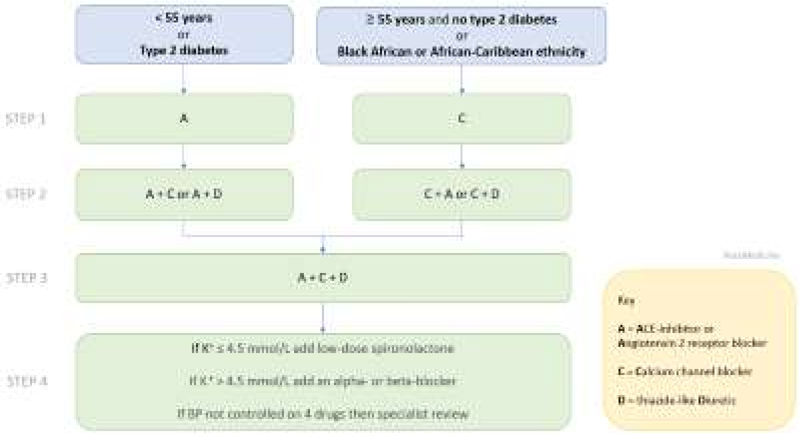
- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day)
    - significantly increases the risk of hypotension

Monitoring

- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
    - a rise in the creatinine and potassium may be expected after starting ACE inhibitors
    - acceptable changes are an increase in serum creatinine, up to 30% from baseline and an increase in potassium up to 5.5 mmol/l.
    - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis

Examples of common ACE inhibitors

- ramipril
- enalapril
- lisinopril


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 53 👎 34

2014 Chronic kidney disease guidelines

BNF 👍 50 👎 25

ACE inhibitors

Suggest link Report broken link

Score: **23.7%**

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Question 39 of 129

A 22-year-old Afro-Caribbean man presents with chest pain. He states the pain is sharp in nature, worse on inspiration, and localised to the right lateral chest wall. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	140ms
QRS	110ms
QTc	420ms
T waves	Inverted in V1-V6
ST segments	No elevation or depression

What is the most likely explanation of these ECG results?

- ○ NSTEMI
- ○ STEMI
- ○ Normal variant
- ○ Prinzmetal's angina (variant angina)
- ○ Stable angina pectoris

Submit answer

Reference ranges

Score: **23.7%**

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Question 39 of 129 ✗

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An ECG is performed:

P waves	Normal morphology
PR interval	140ms
QRS	110ms
QTc	420ms
T waves	Inverted in V1-V6
ST segments	No elevation or depression

What is the most likely explanation of these ECG results?

NSTEMI	10%
STEMI	1%
Normal variant	55%
Prinzmetal's angina (variant angina)	31%
Stable angina pectoris	3%

Widespread T wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity

Important for me Less important

Coronary artery disease would be very unlikely in a man of this age. The absence of ST elevation makes an acute STEMI even more unlikely. T-wave inversion can of course be a feature of an NSTEMI or stable angina, however due to the absence of risk factors, and the patients age, it would be a very unlikely diagnosis.

Prinzmetal's angina due to vasospasm would be a possibility. However this usually presents with ST elevation.

Widespread T-wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity and this would be the most likely diagnosis in a young man with no cardiovascular risk factors.

The patients pain is pleuritic in nature and an alternative diagnosis should therefore be considered (e.g. pneumothorax).

Discuss (8) Improve

Next question >

ECG: normal variants

The following ECG changes are considered normal variants in an athlete:

- sinus bradycardia
- junctional rhythm
- first degree heart block
- Mobitz type 1 (Wenckebach phenomenon)

+ Q 123

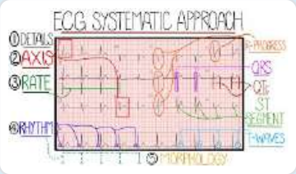
Next question >

Textbooks

High-yield textbook

Extended textbook

Media


ECG SYSTEMATIC APPROACH
①DETAILS
②AXIS
③RATE
④RHYTHM
⑤MORPHOLOGY
R-PROGRESS
QRS
QTc
ST SEGMENT
T-WAVES

ECG Interpretation Made Easy

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **23.1%**

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Question 40 of 129

A 62-year-old man presents to the medical take with a persistent fever, fatigue, and a new-onset heart murmur. There is no relevant past medical history of note and no allergies.

The patient is clinically septic. He has a respiratory rate of 22/min, pulse of 112/min, blood pressure of 91/47 mmHg, and a temperature of 38.7ºC. Bedside echocardiography reveals vegetation on the mitral valve. Blood cultures are sent and results are awaited.

What is the most appropriate empirical antibiotic regimen in this case?

- ○ Amoxicillin +/- gentamicin
- ○ Benzylpenicillin +/- gentamicin
- ○ Flucloxacillin
- ○ Flucloxacillin + rifampicin + gentamicin
- ○ Vancomycin + gentamicin

Submit answer

Reference ranges

Score: **23.1%**

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Question 40 of 129 ✗

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What is the most appropriate empirical antibiotic regimen in this case?

Amoxicillin +/- gentamicin	61%
Benzylpenicillin +/- gentamicin	12%
Flucloxacillin	10%
Flucloxacillin + rifampicin + gentamicin	8%
Vancomycin + gentamicin	9%

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

Important for me Less important

This patient has presented with infective endocarditis. It is important to note that he has native valves and no organism confirmed on blood cultures as of yet. Therefore, empirical treatment with **amoxicillin +/- gentamicin** should be started.

Benzylpenicillin is indicated in endocarditis confirmed to be of streptococcus origin, with gentamicin to be added depending on sensitivities.

Flucloxacillin is indicated when native valve endocarditis secondary to staphylococci has been confirmed on blood cultures.

Flucloxacillin + rifampicin + gentamicin is indicated in prosthetic valve endocarditis caused by staphylococcus.

Vancomycin + gentamicin is an option for empirical treatment in patients who are penicillin-allergic, however, amoxicillin is preferred where this is not the case.

👍 👎 Discuss (5) Improve

Next question >

Infective endocarditis: prognosis and management

Poor prognostic factors
- *Staphylococcus aureus* infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism
- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current antibiotic guidelines (source: British National Formulary)

Scenario	Suggested antibiotic therapy
Initial blind therapy	Native valve • amoxicillin, consider adding low-dose gentamicin If penicillin allergic, MRSA or severe sepsis • vancomycin + low-dose gentamicin

Scenario	Suggested antibiotic therapy
	If prosthetic valve • vancomycin + rifampicin + low-dose gentamicin
Native valve endocarditis caused by staphylococci	Flucloxacillin If penicillin allergic or MRSA • vancomycin + rifampicin
Prosthetic valve endocarditis caused by staphylococci	Flucloxacillin + rifampicin + low-dose gentamicin If penicillin allergic or MRSA • vancomycin + rifampicin + low-dose gentamicin
Endocarditis caused by fully-sensitive streptococci (e.g. viridans)	Benzylpenicillin If penicillin allergic • vancomycin + low-dose gentamicin
Endocarditis caused by less sensitive streptococci	Benzylpenicillin + low-dose gentamicin If penicillin allergic • vancomycin + low-dose gentamicin

Indications for surgery
- severe valvular incompetence
- aortic abscess (often indicated by a lengthening PR interval)
- infections resistant to antibiotics/fungal infections
- cardiac failure refractory to standard medical treatment
- recurrent emboli after antibiotic therapy

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 24

Infective endocarditis guidelines

Suggest link Report broken link

Media

BACTERIAL VEGETATION
IV DRUG USE
INFECTIVE ENDOCARDITIS
OSLER NODES
JANEWAY LESIONS
DUKE'S CRITERIA

Infective Endocarditis

Rhesus Medicine - YouTube 👍 4 👎 0

Endocarditis

Osmosis - YouTube 👍 3 👎 0

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Score: **22.5%**

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Question 41 of 129

A 65-year-old man presents to his GP for evaluation. He has no acute complaints. He takes amlodipine and chlorothiazide for hypertension and paracetamol as required for back pain. Physical examination reveals a well-appearing man with a heart rate of 78 beats per minute with a regular rhythm and blood pressure of 162/78 mmHg. His JVP is not visible and you note that he has a sustained non-displaced apical impulse. There are no thrills or audible murmurs and his heart sounds are normal. His chest is clear to auscultation, his abdomen is soft, non-tender, and there are no palpable masses or organomegaly, and he has trace pedal oedema. His GP reviews his ECG which was performed earlier that morning.

Which of the following is most likely to be identified on this gentleman's ECG?

○ Third degree heart block with a ventricular escape rhythm

○ Absent P-waves

○ Deep S waves in V1 and V2 and tall R-waves in V5 and V6

○ PR-segment depression and global ST-segment elevation

○ A tall S wave in lead I and Q wave with T wave inversion in lead III

Submit answer

Reference ranges

Score: **22.5%**

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Question 41 of 129

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Which of the following is most likely to be identified on this gentleman's ECG?

Third degree heart block with a ventricular escape rhythm	3%
Absent P-waves	4%
Deep S waves in V1 and V2 and tall R-waves in V5 and V6	84%
PR-segment depression and global ST-segment elevation	3%
A tall S wave in lead I and Q wave with T wave inversion in lead III	6%

During a cardiology clinical examination, a sustained apical impulse is consistent with left ventricular hypertrophy, which can be verified on an ECG by identifying deep S waves in V1 and V2 and tall R-waves in V5 and V6

Important for me Less important

This gentleman presents to his GP for a routine checkup. He has a medical history of hypertension and is on medication for this, but has evidence of elevated blood pressure in clinic today. Furthermore, on physical examination, he has a sustained non-displaced apical impulse, a finding that typically represents left ventricular hypertrophy, which can arise as a consequence of longstanding hypertension especially when it is not well controlled. Features of left ventricular hypertrophy can be identified on an ECG, and most typically include deep S waves in V1 and V2 and tall R-waves in V5 and V6.

Third-degree heart block with a ventricular escape rhythm typically results in significant bradycardia. This patient has a normal heart rate and so is unlikely to have a complete heart block.

Absent P-waves is a feature of atrial fibrillation which can be detected on a clinical exam as an irregularly irregular pulse. This gentleman has a regular rhythm and is therefore unlikely to have atrial fibrillation.

PR-segment depression and global 'saddle-shaped' ST-segment elevation are findings consistent with acute pericarditis, which typically presents with pleuritic chest pain worse when lying flat, and relieved when sitting forward. This patient has no symptoms at present and so is unlikely to have acute pericarditis.

A tall S wave in lead I and Q wave with T wave inversion in lead III, so-called 'S1 Q3 T3' is a finding associated with an acute pulmonary embolus. This patient is asymptomatic and is unlikely to have a pulmonary embolus.

👍 👎 Discuss (5) Improve

Next question >

ECG: atrial and ventricular hypertrophy ★

Left ventricular hypertrophy

- sum of S wave in V1 and R wave in V5 or V6 exceeds 40 mm

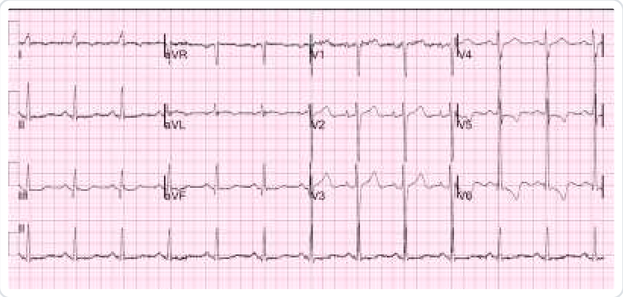
Right ventricular hypertrophy

Left atrial enlargement

- bifid P wave in lead II with a duration > 120 ms

- in V1 the P wave has a negative terminal portion

Right atrial enlargement
- tall P waves in both II and V1 which exceed 0.25 mV



I
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123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **22%**

1 ✗
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3 ✗
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40	✗

41 ✗

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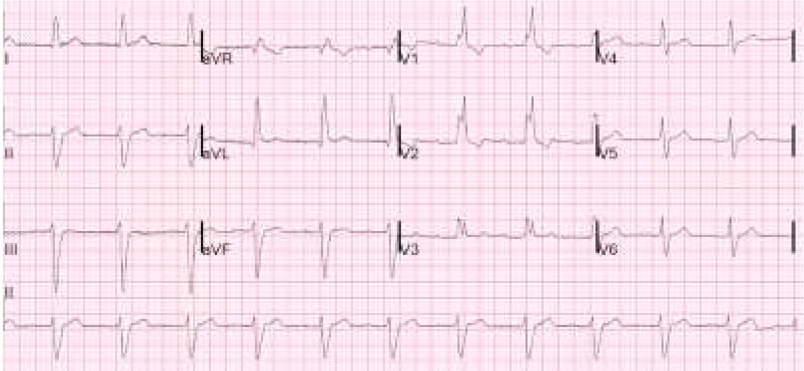
Question 42 of 129

A 77-year-old man is reviewed in the cardiology. He presented to his GP with intermittent dizziness and reduced exercise tolerance. An ECG accompanies the referral letter:



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What does the ECG show?

○ Left bundle branch block ×

○ Long QT syndrome ×

○ ECG changes consistent with hypokalaemia ×

○ Bifascicular block ×

○ Trifascicular block ×

Submit answer

Reference ranges

Score: **22%**

1	✗
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Question 42 of 129 ✓

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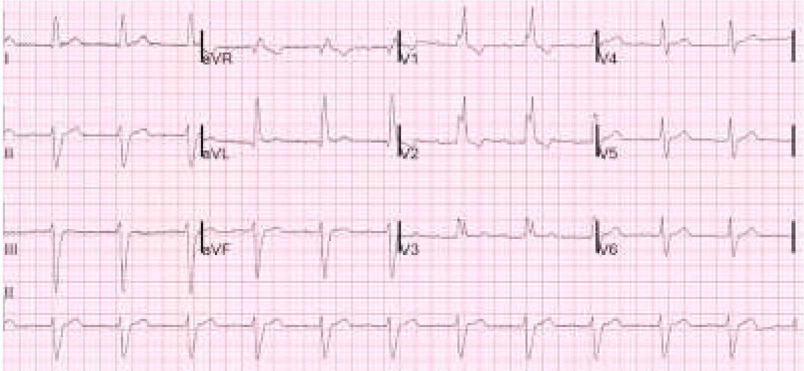


© Image used on license from Dr Smith, University of Minnesota

What does the ECG show?

Left bundle branch block	7%
Long QT syndrome	2%
ECG changes consistent with hypokalaemia	2%
Bifascicular block	28%
Trifascicular block	61%

RBBB +left anterior or posterior hemiblock + 1st-degree heart block = trifascicular block

Important for me   Less important

The ECG demonstrates RBBB + left anterior hemiblock + 1st-degree heart i.e. trifascicular block.

Discuss (17) Improve

Next question

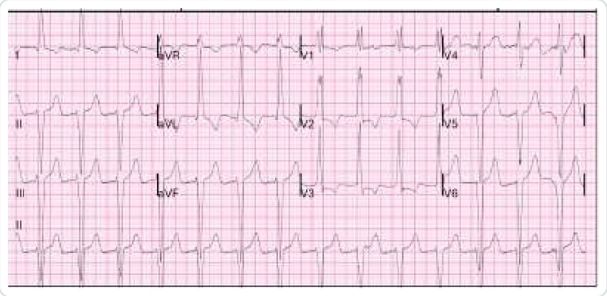
ECG: bi/tri-fascicular block

Bifascicular block

- the combination of RBBB with left anterior or posterior hemiblock
- e.g. RBBB with left axis deviation

Trifascicular block

- features of bifascicular block as above + 1st-degree heart block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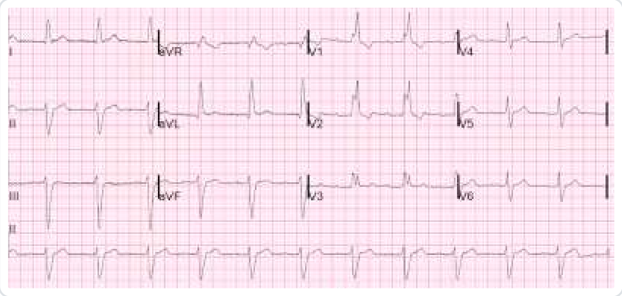
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123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 7 👎 4

Bifascicular block

Life in the fast lane 👍 7 👎 0

Trifascicular block

Suggest link

Report broken link

Score: **23.8%**

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42	✓

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← Question 43 of 129 →

A 56-year-old lady with previously diagnosed rheumatic heart disease with mitral stenosis attends for her annual review. She remains fit and well and can walk 100 metres before she develops breathlessness. She gets occasional burning pains in her chest that come on after rich meals and last 20-30 minutes.

She currently takes aspirin, ramipril and a statin. She does not smoke and is taking regular walks each day. On examination, she has a loud S1 heart sound with a mid-diastolic murmur heard in full expiration when in the left lateral position. Her face has a red flush in the cheeks and there is no JVP visible. Her chest is clear and there is no oedema.

Hb	104 g/l	Na^+	139 mmol/l
Platelets	450 * 10^9/l	K^+	5.2 mmol/l
WBC	5.6 * 10^9/l	Urea	4.5 mmol/l
Neuts	4.5 * 10^9/l	Creatinine	78 µmol/l
Lymphs	0.3 * 10^9/l	CRP	14 mg/l
Troponin	negative		

ECG	sinus rhythm, left ventricular hypertrophy
Chest X-ray	enlarged cardiac shadow with loss of the aorto-pulmonary window
ECHO (today)	mitral valve cross sectional area 0.8cm^2, LV ejection fraction 60%
ECHO (1yr ago)	mitral valve cross sectional area 1.1cm^2, LV ejection fraction 62%

What is the most appropriate intervention?

○ Refer to cardiothoracic surgery ×

○ Furosemide ×

○ Routine follow up with ECHO in 1 year ×

○ Repeat ECHO in 6 months ×

○ Coronary angiogram ×

Submit answer

Reference ranges ﹀

Score: **23.8%**

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Question 43 of 129

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Troponin	negative		

ECG	sinus rhythm, left ventricular hypertrophy
Chest X-ray	enlarged cardiac shadow with loss of the aorto-pulmonary window
ECHO (today)	mitral valve cross sectional area $0.8cm^2$, LV ejection fraction 60%
ECHO (1yr ago)	mitral valve cross sectional area $1.1cm^2$, LV ejection fraction 62%

What is the most appropriate intervention?

Refer to cardiothoracic surgery	53%
Furosemide	2%
Routine follow up with ECHO in 1 year	15%

Repeat ECHO in 6 months 21%

Coronary angiogram 10%

Mitral stenosis with cross sectional area of <1cm2 warrants surgical intervention

Important for me Less important

This lady has known mitral stenosis. She has a valve cross sectional area of less than 1cm ^{2} which is an indication for mitral valve surgery and should be referred immediately. Furosemide would not benefit her as there is no sign of overload. She could continue ECHO surveillance but this will not change her situation. The burning chest pains do not sound cardiac in nature and it would probably be worth trialling omeprazole.

👍 👎 Discuss (6) Improve

Next question >

Mitral stenosis ★

Mitral stenosis describes the obstruction of blood flow across the mitral valve from the left atrium to the left ventricle. This leads to increases in pressure within the left atrium, pulmonary vasculature and right side of the heart.

It is said that the causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever. Rarer causes that may be seen in the exam include mucopolysaccharidoses, carcinoid and endocardial fibroelastosis

Features

- dyspnoea
  - ↑ left atrial pressure → pulmonary venous hypertension
- haemoptysis
  - due to pulmonary pressures and vascular congestion
  - may range from pink frothy sputum to sudden haemorrhage secondary to rupture of thin-walled and dilated bronchial veins
- mid-late diastolic murmur (best heard in expiration)
- loud S1

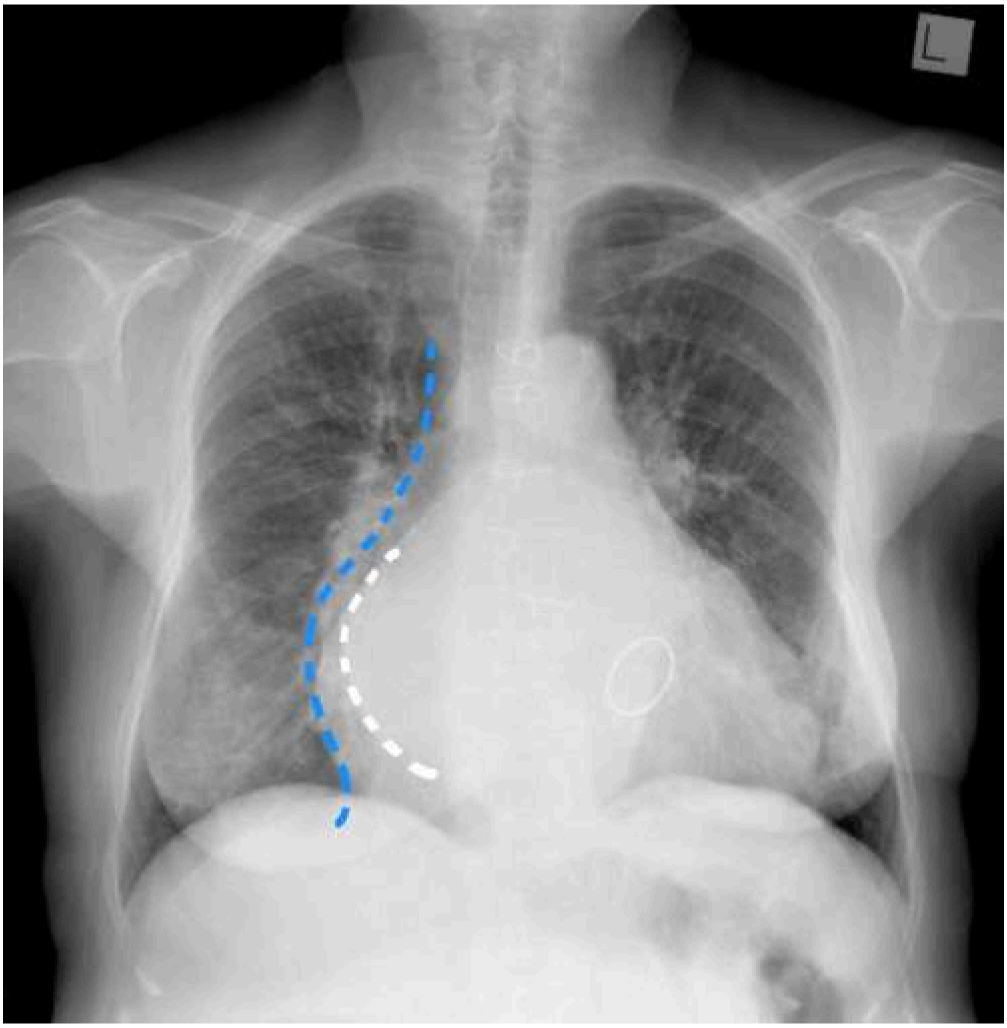
- opening snap
  - indicates mitral valve leaflets are still mobile
- low volume pulse
- malar flush
- atrial fibrillation
  - secondary to ↑ left atrial pressure → left atrial enlargement

Features of severe MS
- length of murmur increases
- opening snap becomes closer to S2

Chest x-ray
- left atrial enlargement may be seen

Echocardiography
- the normal cross-sectional area of the mitral valve is 4-6 sq cm. A 'tight' mitral stenosis implies a cross-sectional area of < 1 sq cm

Management
- patients with associated atrial fibrillation require anticoagulation
  - currently warfarin is still recommended for patients with moderate/severe MS
  - there is an emerging consensus that direct-acting anticoagulants (DOACs) may be suitable for patients with mild MS who develop atrial fibrillation
- asymptomatic patients
  - monitored with regular echocardiograms
  - percutaneous/surgical management is generally not recommended
- symptomatic patients
  - percutaneous mitral balloon valvotomy
  - mitral valve surgery (commissurotomy, or valve replacement)



© Image used on license from Radiopaedia

Chest x-ray from a patient with mitral stenosis. This patient has had a sternotomy and a prosthetic mitral valve. There is splaying of the carina with elevation of the left main bronchus, a double right heart border and cardiomegaly. The features are those of left atrial enlargement. Although the entire heart is enlarged, a double contour is seen through the right side of the heart. The more medial line is the enlarged left atrium (white dotted line) and the heart border is more lateral (blue dotted line).

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 20 👎 11

Mitral valve stenosis

Suggest link Report broken link

Media

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 4 👎 1

Mitral stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 3 👎 2

Report broken media

Score: **23.3%**

1 ✗
2 ✓
3 ✗

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42	✓
43	✗

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Back to top

A 64-year-old gentleman is admitted to the hospital's coronary care unit following an anterior ST-elevation myocardial infarction requiring revascularisation. This was successfully done using two drug eluding stents in his left anterior descending artery. Angiography during the procedure did not indicate any further lesions.

He has been pain free since his percutaneous intervention and his current examination is unremarkable. His observations include a blood pressure of 124/88 mmHg, a heart rate of 62/min, a respiratory rate of 18/min and he is apyrexial.

He has now had an echocardiogram which indicated moderate ventricular dysfunction (ejection fraction 40-42%), with no residual valvular disease.

His ECG currently is in sinus rhythm, with normal PR interval, a QRS complex of 100 ms, and normal QTc.

His bloods are as follows:

Hb	142 g/L	Male: (135-180) Female: (115 - 160)
Platelets	325 * 10^9/L	(150 - 400)
WBC	8.6 * 10^9/L	(4.0 - 11.0)
Na^+	139 mmol/L	(135 - 145)
K^+	3.9 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

He is currently on bisoprolol 5 mg OD, enalapril 2.5 mg BD, aspirin 75 mg OD, ticagrelor 90 mg BD, atorvastatin 80 mg OD and GTN spray PRN.

You are asked by your consultant to optimise his medications/management.

What would be your next step?

- ○ Add ivabradine
- ○ Increase the bisoprolol
- ○ Add isosorbide mononitrate modified release
- ○ Add eplerenone
- ○ Change ticagrelor to clopidogrel

Submit answer

Reference ranges ∨

Score: **23.3%**

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You are asked by your consultant to optimise his medications/management.

What would be your next step?

Add ivabradine	4%
Increase the bisoprolol	11%
Add isosorbide mononitrate modified release	6%
Add eplerenone	74%
Change ticagrelor to clopidogrel	5%

Following myocardial infarction, patients with objective heart failure (moderate to severe left ventricular dysfunction) should be initiated on an ACE inhibitor, a beta-blocker, a statin and a mineralocorticoid receptor antagonist provided no contra-indications exist

Important for me Less important

Given the findings of his echocardiography, one should consider the possibility of addition of a mineralocorticoid receptor antagonist such as eplerenone.

He is currently not symptomatic at all, therefore there is no place in adding isosorbide mononitrate in this patient.

Additionally, given his heart rate one could wonder whether any increase in bisoprolol would render this patient bradycardic.

There is no indication to change his current dual antiplatelet therapy regime.

Ivabradine is a specific inhibition of the pacemaker current, which is used in the management of symptomatic heart failure and ischaemic heart disease that is not managed via the use of typical agents.

👍 👎 Discuss (7) Improve

Next question ›

Myocardial infarction: secondary prevention ★

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2013. Some key points are listed below

All patients should be offered the following drugs:
- dual antiplatelet therapy (aspirin plus a second antiplatelet agent)
- ACE inhibitor
- beta-blocker
- statin

Some selected lifestyle points:
- diet: advise a Mediterranean style diet, switch butter and cheese for plant oil based products. Do not recommend omega-3 supplements or eating oily fish
- exercise: advise 20-30 mins a day until patients are 'slightly breathless'
- sexual activity may resume 4 weeks after an uncomplicated MI. Reassure patients that sex does not increase their likelihood of a further MI. PDE5 inhibitors (e.g, sildenafil) may be used 6 months after a MI. They should however be avoided in patient prescribed either nitrates or nicorandil

Most patients who've had an acute coronary syndrome are now given dual antiplatelet therapy (DAPT). Clopidogrel was previously the second antiplatelet of choice. Now ticagrelor and prasugrel (also ADP-receptor inhibitors) are more widely used. NICE now recommend:
- post acute coronary syndrome (medically managed): add ticagrelor to aspirin, stop ticagrelor after 12 months
- post percutaneous coronary intervention: add prasugrel or ticagrelor to aspirin, stop the second antiplatelet after 12 months
- this 12 month period may be altered for people at a high-risk of bleeding or those who at high-risk of further ischaemic events

Aldosterone antagonists
- patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI

treatment (e.g. eplerenone) should be initiated within 3-14 days of the MI, preferably after ACE inhibitor therapy

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 2017 STEMI guidelines	👍 6 👎 8
Clinical Knowledge Summaries Antiplatelet guidelines	👍 8 👎 4
NICE 2020 Acute coronary syndromes guidelines	👍 16 👎 5

Suggest link Report broken link

Score: **22.7%**

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42 ✓

43 ✗

44 ✗

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Question 45 of 129

A 75-year-old gentleman has recently been diagnosed with heart failure following a myocardial infarction. You review him in clinic. He is currently not complaining of any dyspnoea, orthopnoea or paroxysmal nocturnal dyspnoea and he has no persistent leg oedema. There is no other relevant background medical history.

Examination indicates that he is euvolaemic.

His ECG shows sinus rhythm, Q waves in the inferior leads, a PR interval of 208/msec, normal QRS and no signs of intraventricular conduction delay.

He appears rather anxious and is wondering whether there is anything else that he can do in order to reduce his rate of hospitalisation other than maintaining a healthy lifestyle and taking his medications. He is aware that his brother, who suffers with chronic obstructive pulmonary disease, has a just-in-case box at home and is entitled to vaccinations. He is wondering whether there is any vaccinations or additional antibiotics/medications that could be useful to him.

What would you offer this gentleman?

- ○ Annual pneumococcus vaccination should be offered
- ○ Phenoxymethylpenicillin prescription (prophylactic)
- ○ Annual influenza vaccination
- ○ *Haemophilus influenzae* B vaccination
- ○ Pill-in-the-pocket bumetanide

Submit answer

Reference ranges

Score: **22.7%**

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What would you offer this gentleman?

Annual pneumococcus vaccination should be offered	13%
Phenoxymethylpenicillin prescription (prophylactic)	1%
Annual influenza vaccination	79%
Haemophilus influenzae B vaccination	2%
Pill-in-the-pocket bumetanide	5%

As part of the broad lifestyle approach to heart failure, annual influenza vaccine should be offered

Important for me Less important

Patients with heart failure are encouraged to have the influenza and pneumococcus vaccine, as common respiratory infections are more prevalent in that population and have also the propensity of worsening heart failure and causing decompensation.

The correct answer in this case is annual influenza vaccine; pneumococcus vaccination is usually an once-off regime, provided that the patient is not asplenic or suffering from other chronic diseases.

Haemophilus influenzae B vaccination is advocated in patients with asplenia as they are particularly vulnerable to encapsulated organisms but it has no position in the long-term management of heart failure patients.

Phenoxymethylpenicillin and other antibiotics as prophylaxis are not advocated in heart failure management with regards to reducing infection rates.

There is no strong evidence in supporting a 'pill-in-the-pocket' strategy in patients with heart failure, however patients are encouraged to be aware of the potential manipulation needed when it comes to diuretic therapy and when to seek urgent medical care.

👍 👎 Discuss (2) Improve

Next question ›

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**

- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**
- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

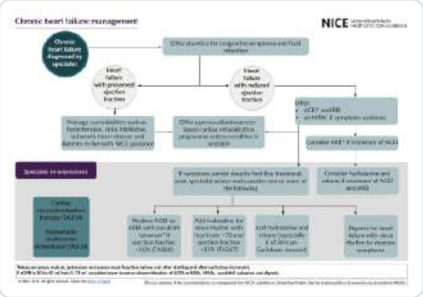
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy
- ivabradine
  - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
  - criteria: left ventricular fraction < 35%
  - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
  - should be initiated following ACEi or ARB wash-out period

- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question

Textbooks

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Links

NICE 👍 14 👎 13

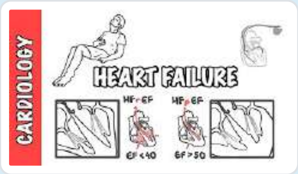
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

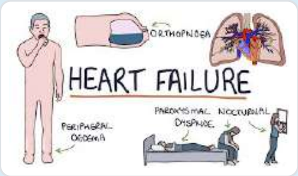
Suggest link   Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PERIPHERAL OEDEMA
PAROXYSMAL NOCTURNAL DYSPNOEA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **22.2%**

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Question 46 of 129

A 31-year-old woman is brought into the Emergency Department by her husband. He tells you that she is abnormally drowsy and he has difficulty rousing her. She has a background of depression which is medicated by her General Practitioner, but is otherwise well.

On examination she is afebrile but has dry, warm skin. Her heart rate is 140bpm with a blood pressure of 105/90mmHg. She has a respiratory rate of 8 breaths per minute and saturating at 92% on air. Her Glasgow coma scale (GCS) is 12 (E3V4M5) and she appears agitated. Her pupils are dilated and she has hyperreflexia. Whilst examining her abdomen it is noted that she has a palpable bladder and bowel sounds are absent.

Investigations:

Arterial blood gas (ABG):

pH	7.29	(7.35-7.45)
PCO2	7.2 kPa	(4.9-6.1 kPa)
PO2	10.1 kPa	(10-13.1 kPa)
Bicarbonate (HCO3-)	28 mmol/L	(22-28 mmol/L)
Base Excess (BE)	3.1 mmol/L	(-2 to 2 mmol/L)
Sodium	134 mmol/L	(135-145 mmol/L)
Potassium	4.4 mmol/L	(3.5-5.5 mmol/L)
Chloride	105 mmol/L	(95-110 mmol/L)
Lactate	2.1 mmol/L	(0.2-1.6 mmol/L)
Glucose	5.3 mmol/L	(4-7 mmol/L)

ECG - Polymorphic ventricular tachycardia, QTc 500ms.

What is the most important next step in the management of this patient?

○ Synchronised DC cardioversion ×

○ Intravenous amiodarone ×

○ Intravenous magnesium sulphate ×

○ Intravenous lidocaine ×

○ Intravenous sodium bicarbonate ×

Submit answer

Reference ranges

HTTPS://T.ME/+AOWEXXQPZDM1NJZK

Score: **22.2%**

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Question 46 of 129

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Glucose	5.3 mmol/L	(4-7 mmol/L)

ECG - Polymorphic ventricular tachycardia, QTc 500ms.

What is the most important next step in the management of this patient?

Synchronised DC cardioversion 10%

Intravenous amiodarone 3%

Intravenous magnesium sulphate	69%
Intravenous lidocaine	1%
Intravenous sodium bicarbonate	17%

IV magnesium sulfate is used to treat torsades de pointes

Important for me Less important

This woman has had a tricyclic antidepressant (TCA) overdose. In excess, TCAs can cause QT interval prolongation which can predispose to ventricular arrhythmias. A polymorphic ventricular arrhythmia with a prolonged QT interval is characteristic of torsades de pointes. The treatment for this is intravenous magnesium sulphate.

As she currently has a stable blood pressure (systolic > 90mmHg), DC cardioversion would not be appropriate at this stage. Intravenous amiodarone and lidocaine can be used to medically cardiovert a monomorphic ventricular tachycardia, but are not the treatment of choice in torsades de pointes.

Sodium bicarbonate is the first-line treatment of TCA overdose. However, in the presence of torsades de pointes, intravenous magnesium sulphate is the more appropriate choice to give initially.

👍 👎 Discuss (5) Improve

Next question >

Torsades de pointes ★

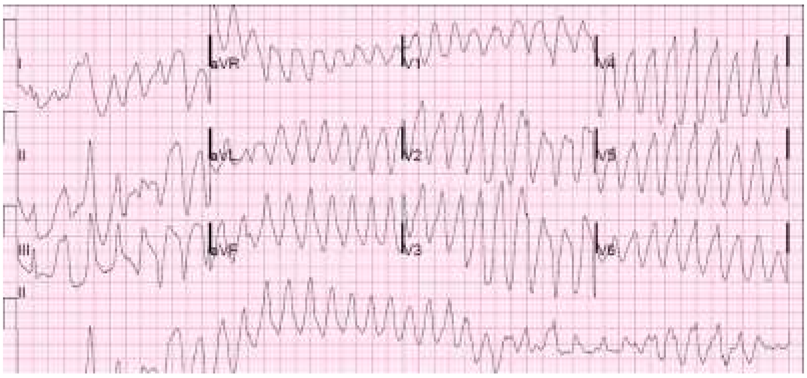
Torsades de pointes ('twisting of the points') is a form of polymorphic ventricular tachycardia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death.

Causes of long QT interval
- congenital
    - Jervell-Lange-Nielsen syndrome
    - Romano-Ward syndrome

- antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs
- tricyclic antidepressants
- antipsychotics
- chloroquine
- terfenadine
- erythromycin
- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia
- myocarditis
- hypothermia
- subarachnoid haemorrhage

Management

- IV magnesium sulphate



© Image used on license from Dr Smith, University of Minnesota

ECG showing torsades de pointes

123

Next question

Textbooks

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Links

Life in the Fast Lane 👍 4 👎 0

Torsades de pointes

Suggest link Report broken link

Media

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 3 👎 1

Report broken media

Score: **21.7%**

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A 72-year-old man attends the emergency department with mild epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 80 beats per minute, blood pressure 145/85 mmHg, respiratory rate 14 breaths per minute, SpO2 96% (on air), and temperature 37.5ºC.

Blood results are as follows:

Hb	144 g/L	Male: (135-180) Female: (115 - 160)
Platelets	72 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)

Prothrombin time (PT)	114 secs	(10-14 secs)
INR	10.2	(<1.1)
Activated partial thromboplastin time (APTT)	54 secs	(25-35 secs)
Fibrinogen	3.4 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

○ Fresh frozen plasma

● Intravenous 10mg vitamin K

○ Intravenous 3mg vitamin K

○ Platelet transfusion

○ Prothrombin complex concentrate

Submit answer

31	✗
32	✗
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Question 47 of 129 ✗

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INR	10.2	(<1.1)
Activated partial thromboplastin time (APTT)	54 secs	(25-35 secs)
Fibrinogen	3.4 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

Fresh frozen plasma	3%
Intravenous 10mg vitamin K	27%
Intravenous 3mg vitamin K	58%
Platelet transfusion	0%
Prothrombin complex concentrate	13%

INR > 8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, repeat dose of vitamin K if INR high after 24 hours, restart when INR < 5.0

Important for me Less important

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR >8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0.

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In warfarin-associated major haemorrhages, FFP should only be given if prothrombin complex concentrate is unavailable.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard transfusion trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100. The patient in this clinical case has a platelet count of 72 and thus does not require a platelet transfusion.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation within minutes of administration however administration of vitamin K is also required to counteract the long half-life of warfarin.

👍 👎 Discuss (1) Improve

Next question >

Warfarin: management of high INR

The following is based on the BNF guidelines, which in turn take into account the British Committee for Standards in Haematology (BCSH) guidelines.

Situation	Management
Major bleeding (e.g. variceal haemorrhage, intracranial haemorrhage)	Stop warfarin Give intravenous vitamin K 5mg Prothrombin complex concentrate - if not available then FFP*
INR > 8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Repeat dose of vitamin K if INR still too high after 24 hours Restart warfarin when INR < 5.0
INR > 8.0 **No bleeding**	Stop warfarin Give vitamin K 1-5mg by mouth, using the intravenous preparation orally Repeat dose of vitamin K if INR still too high after 24 hours Restart when INR < 5.0
INR 5.0-8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Restart when INR < 5.0
INR 5.0-8.0 **No bleeding**	Withhold 1 or 2 doses of warfarin Reduce subsequent maintenance dose

*as FFP can take time to defrost prothrombin complex concentrate should be considered in cases of intracranial haemorrhage

+ Q 123

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BNF 👍 16 👎 11

Warfarin

BNF 👍 18 👎 10

Oral anticoagulants

Suggest link

Report broken link

Score: **21.3%**

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Question 48 of 129

A 50-year-old female is admitted on the acute medical take with palpitations. She recalls on-and-off palpitations for over one year, however today they have been ongoing. On examination, she is haemodynamically stable with an irregularly irregular pulse of 140 b.p.m. There is no clinical evidence of heart failure. Her ECG shows atrial fibrillation. Which of the following medical conditions is a contra-indication to initiating beta-blocker therapy in this patient?

- ○ Intercurrent treatment with dihydropyridine calcium-channel blockers
- ○ Phaeochromocytoma on phenoxybenzamine
- ○ Sick sinus syndrome with a permanent pacemaker
- ○ Second-degree heart block
- ○ Mild peripheral vascular disease

Submit answer

Reference ranges

Score: **21.3%**

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Intercurrent treatment with dihydropyridine calcium-channel blockers	23%
Phaeochromocytoma on phenoxybenzamine	9%
Sick sinus syndrome with a permanent pacemaker	16%
Second-degree heart block	50%
Mild peripheral vascular disease	2%

Given the ubiquity of beta-blockers in medical practice it is important to know contra-indications for their use.

Although the most commonly cited contraindication to beta-blocker therapy is asthma / COPD, there is increasing evidence that in the latter cohort, cardioselective beta-blockers (atenolol, bisoprolol, metoprolol) are well-tolerated with a favourable safety profile.

Beta-blockers slow conduction at the AV node and therefore should be avoided in AV nodal disease as they can lead to complete heart block. Thus they should be avoided in second-degree heart block.

Beta-blockers also inhibit beta1-adrenoceptor activity at the sinoatrial node (i.e. are negative chronotropes) so are contraindicated in sick sinus syndrome. However, in the presence of a permanent pacemaker (PPM), beta-blocker use is safe.

Due to the synergistic effect of beta- and calcium-channel blockade on myocardial excitability, non-dihydropyridine calcium channel blockers (diltiazem and verapamil) should not be co-administered with beta blockers.

An untreated phaeochromocytoma causes increased alpha- and beta-adrenergic activity. Thus the administration of a beta-blocker alone will lead to unopposed alpha-agonism which can lead to vasoconstriction and refractory hypertension. However, if a patient is taking an alpha-blocking agent (e.g. phenoxybenzamine), then beta-blockers may also be initiated.

👍 👎 Discuss (5) Improve

Next question >

Beta-blockers ★

Beta-blockers are an important class of drug used mainly in the management of cardiovascular disorders.

Indications
- angina
- post-myocardial infarction
- heart failure: beta-blockers were previously avoided in heart failure but there is now strong evidence that certain beta-blockers improve both symptoms and mortality
- arrhythmias: beta-blockers have now replaced digoxin as the rate-control drug of choice in atrial fibrillation
- hypertension: the role of beta-blockers has diminished in recent years due to a lack of evidence in terms of reducing stroke and myocardial infarction.
- thyrotoxicosis
- migraine prophylaxis
- anxiety

Examples
- atenolol
- propranolol: one of the first beta-blockers to be developed. Lipid soluble therefore crosses the blood-brain barrier

Side-effects

- bronchospasm
- cold peripheries
- fatigue
- sleep disturbances, including nightmares
- erectile dysfunction

Contraindications

- uncontrolled heart failure
- asthma
- sick sinus syndrome
- concurrent verapamil use: may precipitate severe bradycardia

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

BNF 👍 21 👎 7

Beta-blockers

Suggest link Report broken link

Media

Adrenergic Antagonists

Alpha and beta blockers

Speed Pharmacology - YouTube 👍 2 👎 1

Report broken media

Score: **20.8%**

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Question 49 of 129

A 57-year-old man with a 2 year history of hypertension is referred for further evaluation due to his blood pressure being difficult to control. The following results were obtained prior to commencing medications:

Na^+	148 mmol/l
K^+	3.2 mmol/l
Creatinine	130 µmol/l
Renin	102 (7-50 IU/mL ambulatory)
Aldosterone	1105 (N: 80-800 ng/dL ambulatory)
Renin:Aldosterone Ratio	10.8 (< 500)
Plasma Metanephrines	0.35 (<0.50 nmol/L)

These results are most consistent with which of the following:

○ Congenital Adrenal Hyperplasia ×

○ Phaeochromocytoma ×

○ Cushing's disease ×

○ Renovascular disease ×

○ Primary hyperaldosteronism ×

Submit answer

Reference ranges

Score: **20.8%**

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47	✗
48	✗
49	-

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A 57-year-old man with a 2 year history of hypertension is referred for further evaluation due to his blood pressure being difficult to control. The following results were obtained prior to commencing medications:

Na^+	148 mmol/l
K^+	3.2 mmol/l
Creatinine	130 µmol/l
Renin	102 (7-50 IU/mL ambulatory)
Aldosterone	1105 (N: 80-800 ng/dL ambulatory)
Renin:Aldosterone Ratio	10.8 (< 500)
Plasma Metanephrines	0.35 (<0.50 nmol/L)

These results are most consistent with which of the following:

Congenital Adrenal Hyperplasia	6%
Phaeochromocytoma	2%
Cushing's disease	5%
Renovascular disease	51%
Primary hyperaldosteronism	36%

The key underlying diagnosis is renal artery stenosis. The key is that the renin-aldosterone ratio is normal. If it was Conn's disease, or primary hyperaldosteronism the renin would be suppressed. The plasma metanephrines indicate that no pheochromocytoma is present as the sensitivity of plasma metanephrines for a pheochromocytoma approaches 100%.

Discuss (10) Improve

Next question ›

Hypertension: secondary causes ★

It is thought that between 5-10% of patients diagnosed with hypertension have **primary hyperaldosteronism**, including Conn's syndrome. This makes it the single most common cause of secondary hypertension.

Renal disease accounts for a large percentage of the other cases of secondary hypertension. Conditions which may increase the blood pressure include:
- glomerulonephritis
- pyelonephritis
- adult polycystic kidney disease
- renal artery stenosis

Endocrine disorders (other than primary hyperaldosteronism) may also result in increased blood pressure:
- phaeochromocytoma
- Cushing's syndrome
- Liddle's syndrome
- congenital adrenal hyperplasia (11-beta hydroxylase deficiency)
- acromegaly

Drug causes:
- steroids
- monoamine oxidase inhibitors
- the combined oral contraceptive pill
- NSAIDs
- leflunomide

Other causes include:
- pregnancy
- coarctation of the aorta

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Media

Hypertension

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **20.4%**

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48 ✗
49 ✗

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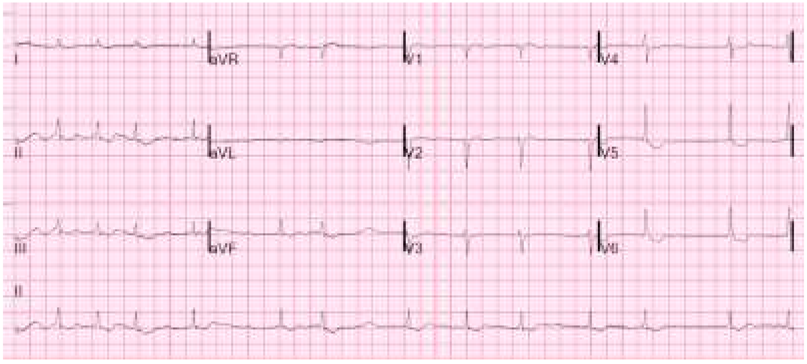
A 77-year-old woman is seen in a heart failure clinic. Her past medical history includes congestive cardiac failure, atrial fibrillation, hypertension, and depression. Her regular medications include bisoprolol, digoxin, amlodipine, furosemide, and citalopram.

Repeat digoxin levels are displayed below:

Digoxin level	1.1 ng/mL	(0.7-2.0)



© Image used on license from Dr Smith, University of Minnesota

Which agent is responsible for the abnormalities displayed in her ECG?

- ○ Amlodipine ×
- ○ Bisoprolol ×
- ○ Citalopram ×
- ○ Digoxin ×
- ○ Furosemide ×

Submit answer

Reference ranges

35	✗
36	✗
37	✗
38	✗
39	✗
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41	✗
42	✓
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46	✗
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48	✗
49	✗
50	-

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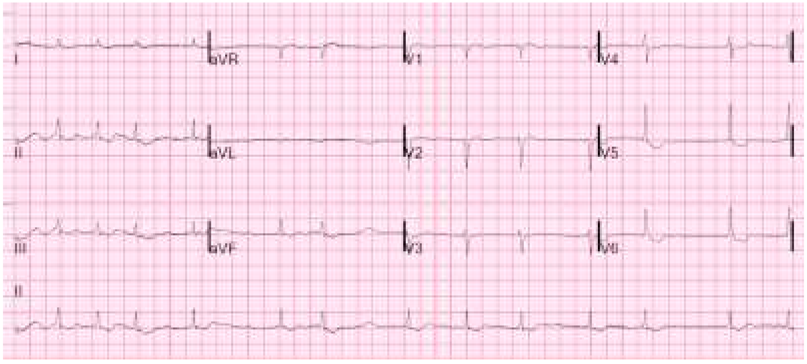
Question 50 of 129 ✗

A 77-year-old woman is seen in a heart failure clinic. Her past medical history includes congestive cardiac failure, atrial fibrillation, hypertension, and depression. Her regular medications include bisoprolol, digoxin, amlodipine, furosemide, and citalopram.

Repeat digoxin levels are displayed below:

Digoxin level	1.1 ng/mL	(0.7-2.0)



© Image used on license from Dr Smith, University of Minnesota

Which agent is responsible for the abnormalities displayed in her ECG?

Amlodipine	1%
Bisoprolol	9%
Citalopram	27%
Digoxin	51%
Furosemide	11%

This woman's ECG displays 'scooped' ST depression in leads II, III, aVF, V5, and V6 in the context of atrial fibrillation. This is a classical sign seen in the digoxin effect, the ECG changes that arise with digoxin within normal therapeutic levels. It is important to note that in digoxin toxicity, the most common dysrhythmias are supraventricular tachycardia with a slow ventricular response due to blocking of the AV node.

Whilst calcium channel blockers can significantly disrupt the ECG, this is associated primarily with non-dihydropyridines such as verapamil or diltiazem, rather than dihydropyridines such as amlodipine which are less cardioselective.

Bisoprolol can give rise to bradycardia and PR prolongation on ECG but the ST changes observed here are consistent with the digoxin effect. Therefore, bisoprolol is incorrect.

Citalopram is a selective serotonin reuptake inhibitor and therefore can result in QT prolongation. The ST changes as described here are not consistent with this syndrome and therefore this is an incorrect answer.

Furosemide is a loop diuretic that can result in hypokalemia and therefore flattened and inverted T waves. The ST segment in this ECG shows the classical 'scooped' appearance and therefore is more consistent with the digoxin effect rather than hypokalemia. Furosemide is therefore incorrect.

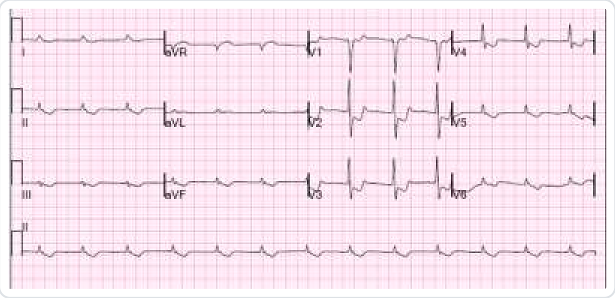
👍 👎 Discuss (2) Improve

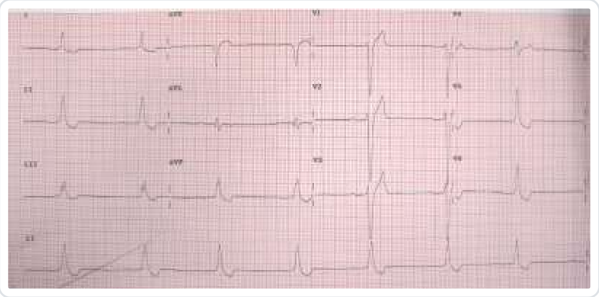
Next question >

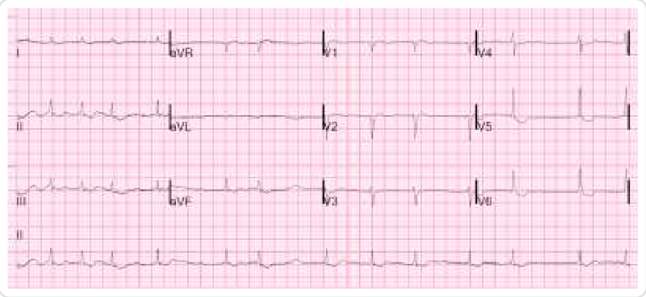
ECG: digoxin

ECG features

- down-sloping ST depression ('reverse tick', 'scooped out')
- flattened/inverted T waves
- short QT interval
- arrhythmias e.g. AV block, bradycardia







I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

AI + Q 123

Next question ›

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 18 3

ECG Features demonstrating the Digoxin Effect

Suggest link Report broken link

Score: **20%**

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Question 51 of 129

An 82-year-old man sees his GP to discuss his medication. He complains of shortness of breath on minimal exertion and says that he now has to sleep upright in his armchair. His medication includes lisinopril, metoprolol, spironolactone, furosemide, aspirin, simvastatin, nifedipine and metformin.

Physical examination shows a heart rate of 69 beats per minute with a regular rhythm and blood pressure of 118/100 mmHg. His peripheries are cool, and his carotid pulse is difficult to palpate. He has a sustained apical impulse palpable in the 5th intercostal space in the mid-clavicular line. He has a palpable thrill in the second intercostal space at the right sternal border where his GP hears a loud crescendo-decrescendo murmur which radiates into his neck. He has a quiet S1, an inaudible S2, and a loud S4. His chest is clear to auscultation and he is noted to have 1+ peripheral oedema.

Which of the following physical examination findings are most suggestive of severe aortic stenosis?

○ Loud crescendo-decrescendo murmur

○ Quiet S1

○ Inaudible S2

○ Palpable thrill

○ Narrow pulse pressure

Submit answer

Reference ranges

Score: **20%**

1	✗
2	✓
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Which of the following physical examination findings are most suggestive of severe aortic stenosis?

Loud crescendo-decrescendo murmur	7%
Quiet S1	20%
Inaudible S2	43%
Palpable thrill	9%
Narrow pulse pressure	22%

Aortic stenosis - a soft S2 is a feature of severe disease

Important for me Less important

The feature identified on physical examination that is most suggestive of severe aortic stenosis is an inaudible second heart sound.

The loudness of a murmur does not correlate with its severity. In the case of regurgitant murmurs, for example, quiet murmurs can represent severe regurgitation as there is early equalisation between the two cardiac chambers involved.

A quiet S1 may reflect significant aortic valve calcification but does not necessarily reflect the severity of the stenosis

A palpable thrill and narrow pulse pressure can occur in severe aortic stenosis but are not as indicative of severe aortic stenosis as an absent second heart sound

👍 👎 Discuss (5) Improve

Next question >

Aortic stenosis ★

Clinical features of symptomatic disease
- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur
  - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
  - classically radiates to the carotids
  - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis
- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
    - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
    - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
    - may be used in children with no aortic valve calcification
    - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

Guidelines on the management of valvular heart disease

Suggest link Report broken link

Media

Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1

AORTIC VALVE DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2

AORTIC STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

Report broken media

Score: **19.6%**

1	✗
2	✓
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47	✗
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50	✗
51	✗

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Question 52 of 129

A 71-year-old patient presents to the Emergency Department with a two hour history of crushing central chest pain. He is known to have a history of ischaemic heart disease. The ECG shows the following:

- ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF
- ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2
- ST elevation in V5-V6

Where is the lesion most likely to be?

○ Left anterior descending ×

○ Left circumflex ×

○ Right coronary artery ×

○ Left main stem ×

○ Posterior interventricular ×

Submit answer

Reference ranges

Score: **19.6%**

1 ✗
2 ✓
3 ✗
4 ✗

5	✓
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52	-

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- ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF
- ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2
- ST elevation in V5-V6

Where is the lesion most likely to be?

Left anterior descending	5%
🟩 Left circumflex	35%
Right coronary artery	33%
Left main stem	7%
🟥 Posterior interventricular	19%

Ischaemic changes in leads I, aVL +/- V5-6 - left circumflex

Important for me   Less important

These are classical findings of a circumflex occlusion. The table below shows how the changes correspond to the cardiac anatomy:

ECG changes	Component of infarction
ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF	Inferior component of infarction

ECG changes	Component of infarction
ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2	Posterior component of infarction
ST elevation in V5-V6	Lateral component of infarction

Please see the link for an example ECG with a description of the changes.

👍 👎 Discuss (16) Improve

Next question >

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves	Usually left circumflex, also right coronary

	ECG changes	Coronary artery
	- dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

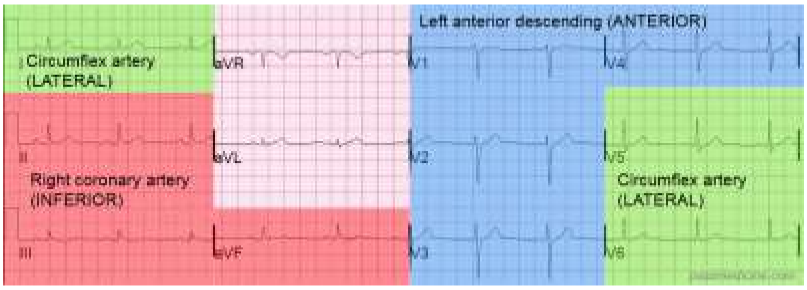
Left anterior descending (ANTERIOR)
Circumflex artery
(LATERAL)
aVR
V1
V4
II
aVL
V2
V5
Right coronary artery
(INFERIOR)
Circumflex artery
(LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

+ Q 123

Next question >

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

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Extended textbook

Links

Postgraduate Medical Journal 👍 7 👎 11

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 2 👎 6

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **19.2%**

1	✗
2	✓
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51	✗
52	✗

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Question 53 of 129

A 24-year-old male presents to the emergency department with a collapse. The previous night he had been out drinking heavily with his friends in a nightclub. A friend attends with him and states that some of the lads had taken MCAT (Mephedrone) but he is unsure whether Michael also took MCAT. Michael felt well when he woke up but realised that he was late for his football match so rushed out of the house without any breakfast. He had just scored the first goal of the match when he collapsed. When he came around he complained of palpitations.

His past medical history includes asthma and hay fever for which he takes regular antihistamines. During the ambulance ride to hospital the paramedic performed some observations and an ECG. He was found to have a blood glucose of 3.6 and was given some GlucoGel and a biscuit. Other observations included a blood pressure of 90/60 mmHg, heart rate of 135/min which was irregular, irregular in character, respiratory rate of 16/min, afebrile.

The emergency department doctor reviewed the ECG and diagnosed atrial fibrillation with a fast ventricular response and prescribed digoxin in view of his asthma history and blood pressure readings. The patient had a normal QTc. Five minutes later the cardiac monitor showed a broad complex tachycardia and Michael became unresponsive.

What was the cause of the patient's collapse?

○ Hypertrophic cardiomyopathy (HCM) ×

○ MCAT related arrhythmia ×

○ Wolff-Parkinson-White syndrome ×

○ Alcohol induced atrial fibrillation ×

○ Torsades de pointes ×

Submit answer

Reference ranges

Score: **19.2%**

1	✗
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53	-

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What was the cause of the patient's collapse?

Hypertrophic cardiomyopathy (HCM)	16%
MCAT related arrhythmia	8%
Wolff-Parkinson-White syndrome	56%
Alcohol induced atrial fibrillation	4%
Torsades de pointes	16%

Wolff-Parkinson-White syndrome (WPW) is a disorder of the conduction system also known as a pre-excitation syndrome. The incidence of WPW is between 0.1% and 0.3% in the general population. Sudden cardiac death in people with WPW is rare (incidence of less than 0.6%).

In WPW there is an abnormal accessory pathway called the 'Bundle of Kent' found between the atria and ventricles. If this pathway is used signals may stimulate the ventricles to contract prematurely, resulting in atrioventricular re-entrant tachycardia.

Normally the AV node acts as a gatekeeper limiting the amount of electrical activity that can reach the ventricles. If a patient has an arrhythmia whereby the atria generate an excessively fast rhythm such as atrial flutter or fibrillation the AV node is able to reduce the amount of electrical activity that reaches the ventricles. However, the Bundle of Kent does not possess the ability to limit the electrical signals to the ventricles. If the person has an atrial arrhythmia the signals can pass down the bundle and cause the ventricles to contract at the same rate. In the extreme heart rates and when the accessory pathway is used the patient may develop dangerous cardiac arrhythmias which can trigger ventricular fibrillation and lead to death.

In WPW certain medications that can block the AV node or those that can enhance conduction down the accessory pathway by increasing the refractory period in the AV node should be avoided. These include digoxin, adenosine, diltiazem, verapamil, other calcium channel blockers and beta blockers. In this case, the patient's WPW was worsened by administration of digoxin and her arrhythmia developed into ventricular fibrillation.

People with WPW are usually asymptomatic. Symptomatic patients may complain of shortness of breath, palpitations, dizziness and syncope. Diagnosis is usually made by ECG as patients may have the characteristic 'delta wave'. Patients may also be found to be in atrial fibrillation or have SVT Treatment can include cardioversion in emergencies otherwise amiodarone. Definitive treatment would be ablation.

👍 👎 Discuss (13) Improve

Next question ›

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

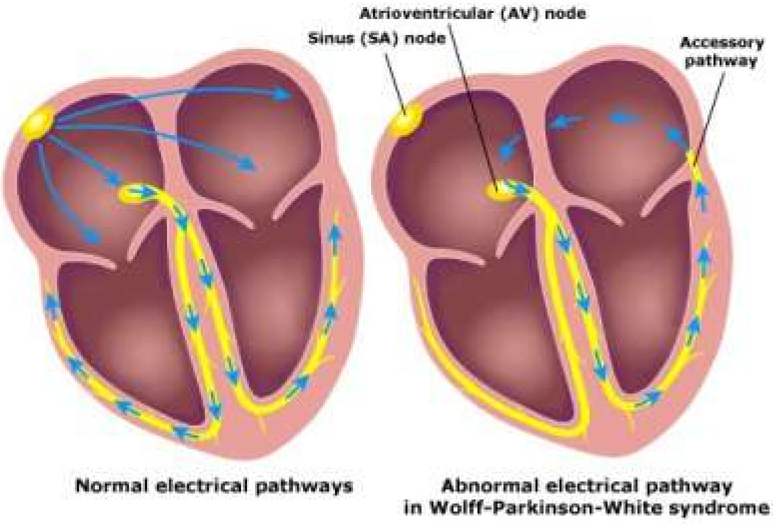
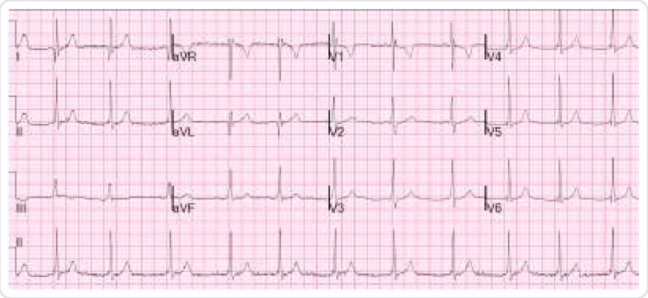

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
  - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway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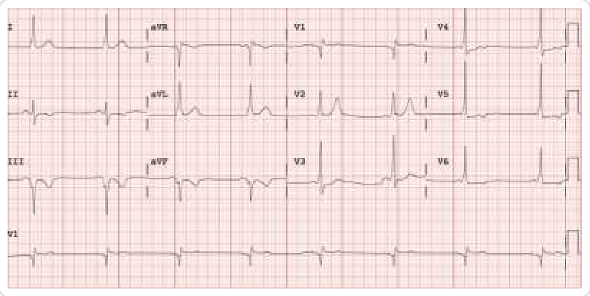

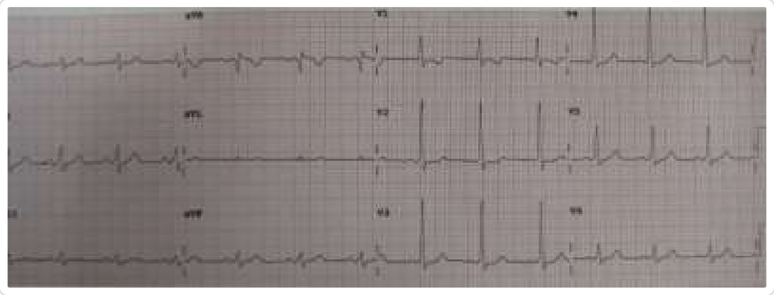

Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
  - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 👍 12 👎 2

Report broken media

Score: **18.9%**

1	✗
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Question 54 of 129

A 65-year-old gentleman is reviewed in cardiology clinic with known cardiomyopathy. He has started to develop symptoms of progressive exertional shortness of breath and is concerned about having this treated. He has not had any other symptoms and on examination, there is no peripheral oedema or chest signs. A recent echocardiogram demonstrates a provoked left ventricular outflow gradient of 64mmHg. What would be the most appropriate medical therapy?

- ○ Phosphodiesterase type 5 inhibitor
- ○ Beta-blocker
- ○ Nitrate
- ○ Digoxin
- ○ Ace-inhibitor

Submit answer

Reference ranges

Score: **18.9%**

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Question 54 of 129

A 65-year-old gentleman is reviewed in cardiology clinic with known cardiomyopathy. He has started to develop symptoms of progressive exertional shortness of breath and is concerned about having this treated. He has not had any other symptoms and on examination, there is no peripheral oedema or chest signs. A recent echocardiogram demonstrates a provoked left ventricular outflow gradient of 64mmHg. What would be the most appropriate medical therapy?

Phosphodiesterase type 5 inhibitor	14%
Beta-blocker	53%
Nitrate	9%
Digoxin	2%
Ace-inhibitor	21%

The correct answer is beta-blocker. Beta-blockers and verapamil can help with symptom management as they control the heart rate to the point where ventricular outflow obstruction is unlikely to occur. Nitrates and ace-inhibitors are contra-indicated as they lower blood pressure which can be very dangerous when combined with hypotension when the outflow is obstructed, whilst phosphodiesterase type 5 inhibitors and digoxin would offer no benefit. Phosphodiesterase type 5 inhibitors have a greater role in pulmonary hypertension whilst digoxin can be used to control the heart rate in AF when the patient is sedentary or there is coexisting heart failure.

Discuss (5) | Improve

Next question

Hypertrophic obstructive cardiomyopathy: management

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500.

Management
- **A**miodarone
- **B**eta-blockers or verapamil for symptoms
- **C**ardioverter defibrillator
- **D**ual chamber pacemaker
- **E**ndocarditis prophylaxis*

Drugs to avoid
- nitrates
- ACE-inhibitors
- inotropes

*although see the 2008 NICE guidelines on infective endocarditis prophylaxis

Next question >

Textbooks

High-yield textbook

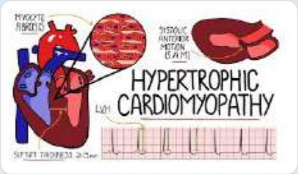
Extended textbook

Media

CARDIOLOGY
HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic obstructive cardiomyopathy

Osmosis - YouTube 👍 8 👎 0


HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic Cardiomyopathy Made Easy

Rhesus Medicine - YouTube 👍 1 👎 0

Hypertrophic cardiomyopathy

Osmosis - YouTube 👍 3 👎 4

Report broken media

Score: **18.5%**

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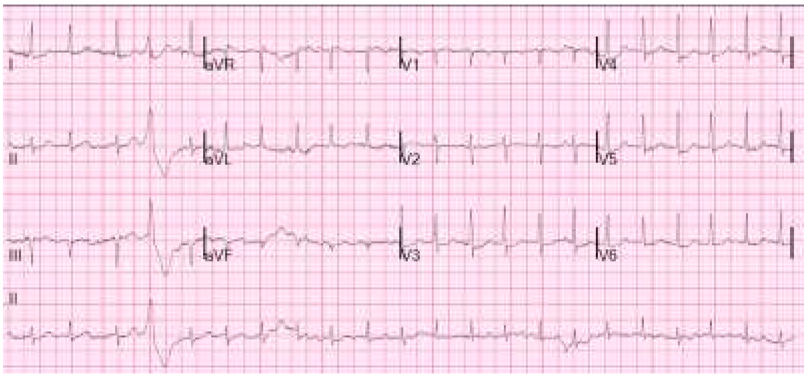
A 54-year-old man presents with a sensation of his heart racing. He has noticed this intermittently over the last several weeks. On examination, his heart sounds are normal with no added sounds. He has a heart rate of 90 beats per minute and a blood pressure of 124/66 mmHg.

His past medical history includes mild asthma only. He is otherwise fit and well and regularly cycles 30 - 40 miles at the weekend. He previously had electrical cardioversion 2 years ago which restored normal sinus rhythm at the time.

His ECG is shown below.



© Image used on license from Dr Smith, University of Minnesota

As well as anticoagulation, what additional medication could be considered before electrical cardioversion?

- ○ Amiodarone
- ○ Aspirin
- ○ Digoxin
- ○ Flecainide
- ○ Ramipril

Submit answer

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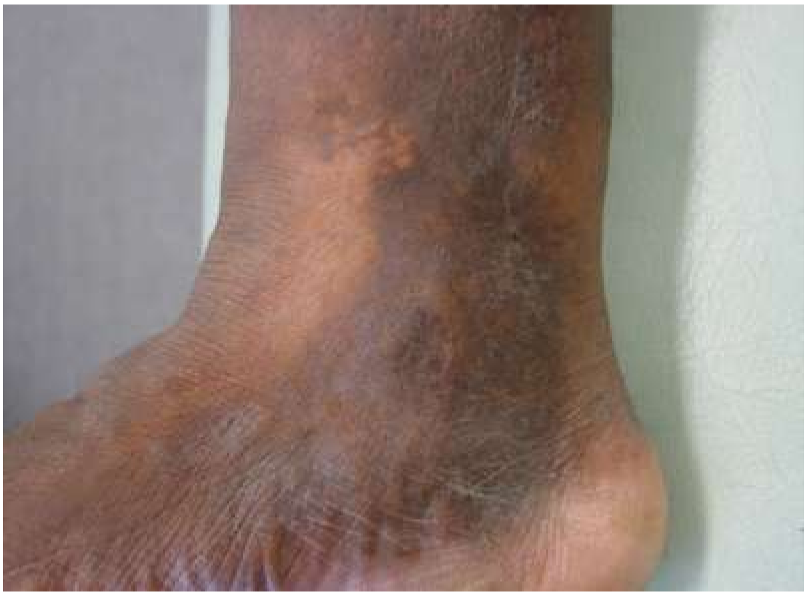
Question 56 of 129

A 69-year-old man visits his General Practitioner complaining of discolouration to his lower legs. He denies itch but complains of intermittent pain that is progressively worsening. He does not visit his doctor regularly and has not had a check-up in several years. He takes no regular medications and chooses to take herbal supplements daily.

Ankle brachial pressure index	1.1

His legs are examined, as shown below:



© Image used on license from DermNet NZ

What is the recommended management for this patient?

- ○ Compression stockings ×
- ○ Low dose aspirin and statin ×
- ○ Oral flucloxacillin ×
- ○ Topical corticosteroids ×
- ○ Topical emollients ×

Submit answer

Reference ranges ⌵

Score: **18.2%**

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Question 56 of 129

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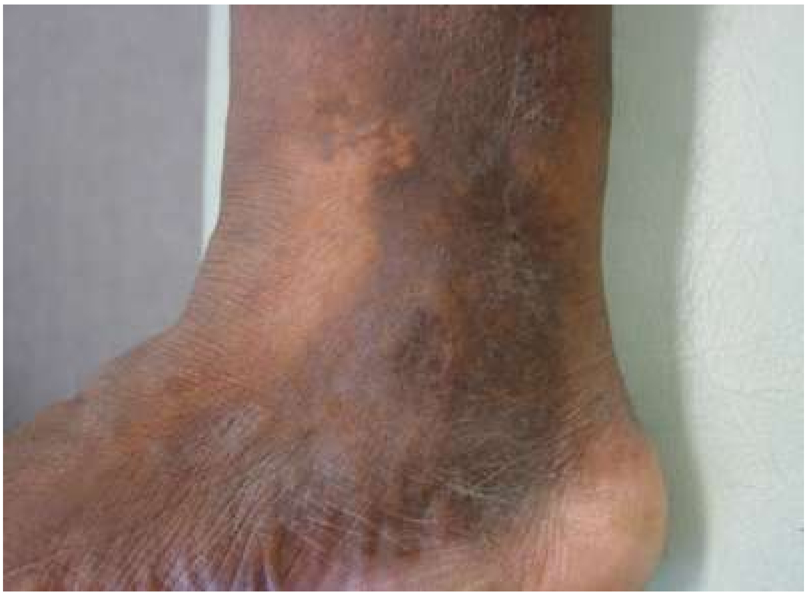


© Image used on license from DermNet NZ

What is the recommended management for this patient?

Compression stockings	55%
Low dose aspirin and statin	34%
Oral flucloxacillin	1%
Topical corticosteroids	3%
Topical emollients	7%

This patient has evidence of discolouration to the lower leg, in keeping with haemosiderin deposition as seen in chronic venous insufficiency. It is likely that this patient has an underlying diagnosis of venous insufficiency and varicose veins. Risk factors for varicose veins include female sex, obesity, pelvic congestion (e.g. in pregnancy) which increases venous pressure in the lower limbs and increasing age. Other changes that can be seen in patients with varicose veins include lipodermatosclerosis (hardening of the skin of the lower limb) and varicose eczema. The management of choice consists of conservative measures in the first instance including exercise, weight loss and **compression stockings**.

Low dose aspirin and statin is incorrect. Although there is a history of pain, the normal ankle brachial pressure index is reassuring that this patient's pain is not secondary to arterial ischaemia. Therefore, aspirin and statin therapy is not indicated.

Oral flucloxacillin is incorrect. This is used in the management of cellulitis which typically appears as an erythematous and inflamed area of skin that is warm and tender to the touch. This is not in keeping with this patient's presentation.

Topical corticosteroids is incorrect. It is used in the management of some inflammatory skin disorders such as eczema, urticaria and psoriasis. However, it plays no role in the management of varicose veins.

Topical emollients is incorrect. This is used in the treatment of dermatitis which is visualised as dry and exfoliative skin that may show signs of excoriations and hyperpigmentation. This is not seen here.

👍 👎 Discuss Improve

Next question >

Varicose veins ★

Varicose veins are dilated, tortuous, superficial veins that occur secondary to incompetent venous valves, allowing blood to flow back, away from the heart. They most commonly occur in the legs due to reflux in the great saphenous vein and small saphenous vein. Whilst extremely common, the vast majority of patients do not require any intervention.

Risk factors

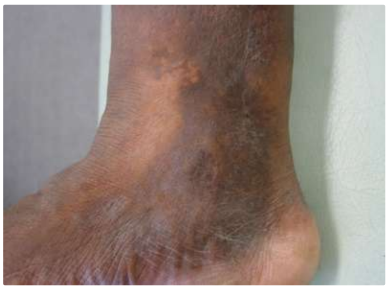
- increasing age
- female gender
- pregnancy
  - the uterus causes compression of the pelvic veins
- obesity

For many patients, the cosmetic appearance may prompt presentation. However other patients may complain of symptoms:

- aching, throbbing
- itching

Other patients may present with complications of varicose veins:

- a variety of skin changes may be seen:
  - varicose eczema (also known as venous stasis)
  - haemosiderin deposition → hyperpigmentation
  - lipodermatosclerosis → hard/tight skin
  - atrophie blanche → hypopigmentation
- bleeding
- superficial thrombophlebitis
- venous ulceration
- deep vein thrombosis



Investigation

- venous duplex ultrasound: this will demonstrate retrograde venous flow

Management

- the majority of patients with varicose veins do not require surgery. Conservative treatments include:

    - leg elevation
    - weight loss
    - regular exercise
    - graduated compression stockings
- reasons for referral to secondary care include:
    - significant/troublesome lower limb symptoms e.g. pain, discomfort or swelling
    - previous bleeding from varicose veins
    - skin changes secondary to chronic venous insufficiency (e.g. pigmentation and eczema)
    - superficial thrombophlebitis
    - an active or healed venous leg ulcer
- possible treatments:
    - endothermal ablation: using either radiofrequency ablation or endovenous laser treatment
    - foam sclerotherapy: irritant foam → inflammatory response → closure of the vein
    - surgery: either ligation or stripping

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries 👍 5 👎 5

Varicose veins

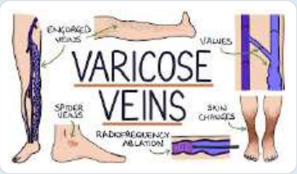
Suggest link Report broken link

Media

SKIN CHANGES
CREAMS
COMPRESSION
CHRONIC VENOUS INSUFFICIENCY
VENOUS HYPERTENSION
ULCERS

Understanding Chronic Venous Insufficiency

Zero To Finals - YouTube 👍 0 👎 0


ENLARGED VEINS
VALVES
VARICOSE VEINS
SPIDER VEINS
SKIN CHANGES
RADIOFREQUENCY ABLATION

Understanding Varicose Veins

Zero To Finals - YouTube 👍 0 👎 0

Varicose veins & chronic venous insufficiency (CVI)

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **17.9%**

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Question 57 of 129

A 79-year-old woman is reviewed in cardiology clinic as part of the ongoing assessment for possible intervention for severe aortic stenosis. The patient had been diagnosed with severe aortic stenosis 4 months previously, after experiencing progressive exertional breathlessness and reduced exercise tolerance. At her initial review at cardiology clinic, the patient had expressed interest in undergoing intervention for aortic stenosis: either surgical aortic valve replacement (SAVR) or transcatheter aortic valve insertion (TAVI). Subsequently, the patient had undergone a variety of investigations to assess her suitability for the above procedures.

The patient had a good functional status, leaving independently with her daughter and still participating in a wide range of community activities. The patient had a long-standing diagnosis of hypertension and had suffered a left cortical stroke 3 years previously. In addition, the patient had chronic obstructive pulmonary disease, although she had stopped smoking 30 years previously. The patient's regular medications were amlodipine, ramipril, clopidogrel, simvastatin and an ipratropium inhaler. The patient used a salbutamol inhaler as required. The patient had no known drug allergies.

A summary of the investigations undergone by the patient is given below.

Haemoglobin	128 g / dL
Mean cell volume	87.1 fl
White cell count	6.3 x 10>3 / microlitre
Platelets	324 x 10>3 / microlitre
Urea	5.3 mmol / L
Creatinine	124 micromol / L
Sodium	136 mmol / L
Potassium	4.6 mmol / L

Investigation	Result
Transthoracic echocardiogram	Severe aortic stenosis (valve area 0.85 cm2); no other valve disease; normal systolic function; no anatomic contraindications to TAVI
Coronary angiography	No evidence of coronary artery disease
Iliofemoral angiography	Severe calcification and tortuosity of iliac arteries; unsuitable for transfemoral TAVI
Pulmonary function tests	Moderate obstructive lung disease

Following the above assessment, surgical aortic valve replacement was estimated to carry a 4.1 % risk of mortality and 3.7 % risk of permanent stroke (intermediate risk). No contraindications to transapical transcatheter aortic valve insertion were identified.

What is the recommended choice of intervention for the patient's aortic stenosis?

○ Surgical aortic valve replacement and transcatheter aortic valve insertion inappropriate

○ Surgical aortic valve replacement with mechanical valve

○ Transapical transcatheter aortic valve insertion

○ Surgical aortic valve replacement with bioprosthetic valve

○ Transfemoral transcatheter aortic valve insertion

Submit answer

Reference ranges

Score: **17.9%**

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Following the above assessment, surgical aortic valve replacement was estimated to carry a 4.1 % risk of mortality and 3.7 % risk of permanent stroke (intermediate risk). No contraindications to transapical transcatheter aortic valve insertion were identified.

What is the recommended choice of intervention for the patient's aortic stenosis?

Surgical aortic valve replacement and transcatheter aortic valve insertion inappropriate	6%
Surgical aortic valve replacement with mechanical valve	9%
Transapical transcatheter aortic valve insertion	42%
Surgical aortic valve replacement with bioprosthetic valve	36%
Transfemoral transcatheter aortic valve insertion	7%

The patient has severe, symptomatic aortic stenosis and without intervention is likely to have a poor prognosis. Given the patient's age, if she was to undergo surgical aortic valve replacement (SAVR) then a bioprosthetic valve would be preferred over a mechanical valve, freeing the patient from the need for lifelong anticoagulation. Typical risk stratification for SAVR defines the estimated risk of peri-operative death as low (< 4 %), intermediate (4-8 %) and high (> 8 %). Due to her comorbidities, the patient has an intermediate risk associated with SAVR, calculated using the STS risk calculator (http://riskcalc.sts.org/stswebriskcalc/#/).

Transcatheter aortic valve insertion (TAVI) was initially developed for patients considered unfit for SAVR due to high predicted mortality. However, a recent randomised controlled trial comparing outcomes for TAVI versus SAVR as an intervention for severe, symptomatic aortic stenosis has demonstrated that transfemoral TAVI can be a suitable intervention for some patients with a low or intermediate risk associated with SAVR. Transfemoral TAVI is increasingly favoured over SAVR as patient age increases.

In contrast, outcomes for transapical TAVI are inferior to SAVR, so this intervention is only appropriate for individuals with an unacceptably high surgical risk. For patients where both SAVR and transapical TAVI are possible, SAVR is strongly favoured at all patient ages.

Therefore, for this patient, bioprosthetic SAVR is the intervention of choice, favoured over transapical TAVI. Transfemoral TAVI is technically impossible in this patient due to the anatomy of her iliac arteries.

Vandvik P, Otto C, Siemieniuk R, Bagur R, Guyatt G, Lytvyn L et al. Choice of intervention for those with severe aortic stenosis. BMJ2016;354:i5085.

👍 | 👎 | Discuss (13) | Improve

Next question >

Aortic stenosis ★

Clinical features of symptomatic disease

- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur
    - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
    - classically radiates to the carotids
    - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
    - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
    - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
    - may be used in children with no aortic valve calcification
    - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

Guidelines on the management of valvular heart disease

Suggest link Report broken link

Media

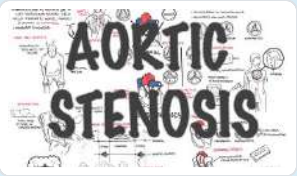
Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1

AORTIC VALVE DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2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STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

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Score: **17.5%**

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Question 58 of 129

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking their secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

- Heart rate 55 beats per minute
- Blood pressure 134/80 mmHg
- Respiratory rate 16 breaths per minute
- Oxygen saturations 98% on air
- Temperature 36.2 °C

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

○ Arrange for urgent repeat PCI ×

○ Arrange for urgent pacemaker insertion ×

○ No additional management is required ×

○ Stop the patient's beta blocker and continue all other medications ×

○ Urgent atropine ×

Submit answer

Reference ranges

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Question 58 of 129

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- Oxygen saturations 98% on air
- Temperature 36.2 ºC

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

Arrange for urgent repeat PCI	1%
Arrange for urgent pacemaker insertion	15%
No additional management is required	69%
Stop the patient's beta blocker and continue all other medications	15%
Urgent atropine	1%

Accelerated idioventricular rhythm is common and unconcerning following recent MI

Important for me Less important

AIVR is a benign ectopic rhythm of ventricular origin. It is common post-MI following the reperfusion of an ischaemic myocardium. AIVR is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable with a normal heart rate and blood pressure.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

Pacemaker insertion is not required as AIVR is due to a benign ectopic rhythm and therefore pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

👍 | 👎 | 💬 Discuss (1) | Improve

Next question >

Accelerated idioventricular rhythm ★

Accelerated idioventricular rhythm (AIVR) is a benign ectopic rhythm of ventricular origin. It usually occurs following the reperfusion of an ischaemic myocardium. It has a rate of 50-110 beats per minute, which helps differentiate it from ventricular bradycardia or ventricular tachycardia.

Pathophysiology:

- Reperfusion of ischaemic tissue, electrolyte abnormalities or drug toxicity leads to an increased depolarisation rate of ventricular myocytes.
- When this depolarisation rate is faster than the rate produced by the sinoatrial node, it becomes the overriding rhythm. Therefore, sinus

bradycardia by vagal excess or reduced sympathetic activity can precipitate AIVR.

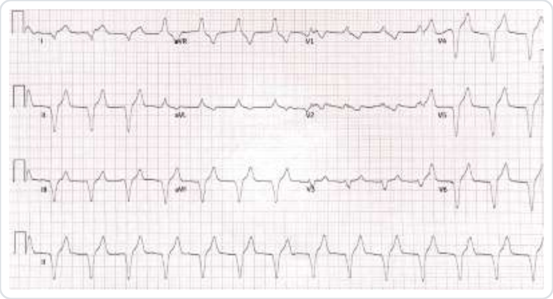
AIVR has a rate of between 50 -110 beats per minute and, therefore, the patient tends to be haemodynamically stable. However, if the rate is fast or there is extensive damage to the myocardium, the patient can become unstable.

Causes:

- Reperfusion following myocardial infarction - by far the most common cause
- Beta-sympathomimetics (e.g. adrenaline)
- Drug toxicity (digoxin/cocaine)
- Electrolyte imbalance
- Cardiomyopathy, congenital heart disease, myocarditis

AIVR is diagnosed based on electrocardiography (ECG) findings:

- AIVR has a gradual onset and termination as the ventricular rate is only slightly faster than sinus rhythm. This may result in ventricular fusion beats on ECG.
- Atrioventricular dissociation - P waves present but not associated with QRS complexes.
- The ECG will show wide QRS complexes >120ms.
- Rate between 50-110 beats per minute.



Treatment:

- AIVR is usually self-limiting and therefore treatment is not necessary, however, occasionally atropine can be used to increase the sinus rate to overcome AIVR.

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **17.2%**

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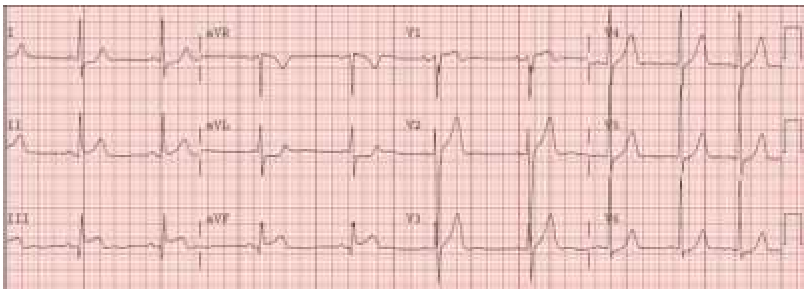
Question 59 of 129

A middle-aged man is admitted to the resuscitation room of the Emergency Department with crushing central chest pain. His ECG is shown below:



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Where is the lesion most likely to be?

○ Left circumflex ×

○ Right coronary ×

○ Left anterior descending ×

○ Posterior interventricular ×

○ Left main stem ×

Submit answer

Reference ranges

Score: **17.2%**

1 ✗

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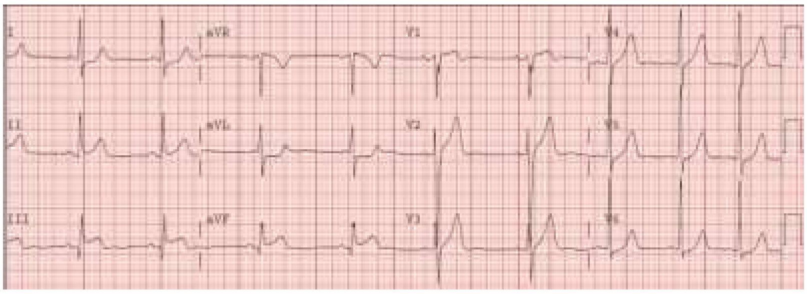
Question 59 of 129 ✗

A middle-aged man is admitted to the resuscitation room of the Emergency Department with crushing central chest pain. His ECG is shown below:



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Where is the lesion most likely to be?

Left circumflex	5%
Right coronary	78%
Left anterior descending	10%
Posterior interventricular	2%
Left main stem	4%

Ischaemic changes in leads II, III, aVF - right coronary

Important for me Less important

There is inferior ST elevation (II, III and aVF) suggestive of a right coronary lesion. Some reciprocal ST depression in I and aVL is also seen.

Discuss (7) Improve

Next question >

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

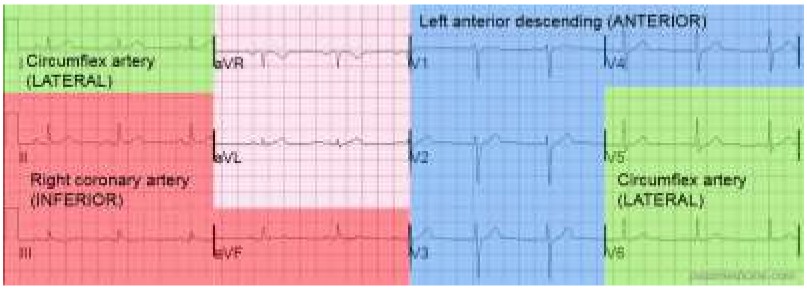
Circumflex artery
(LATERAL)
aVR
Left anterior descending (ANTERIOR)
V1
V4
II
aVL
V2
V5
Right coronary artery
(INFERIOR)
Circumflex artery
(LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

＋ Q 123

Next question ›

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 7 👎 11

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 2 👎 6

Classic circumflex infarction ECG

Suggest link

Report broken link

Score: **16.9%**

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A 14-year-old female presented to her GP having collapsed at school. Her friends stated that whilst she was playing hockey she inexplicably collapsed and had lost consciousness for a few seconds. This was her first episode of collapse. No seizure activity was observed, and she made a full and spontaneous recovery a few moments later. She denied the presence of prodromal symptoms, and other than occasional palpitations upon exertion she was otherwise well. She had a cochlear implant inserted in early childhood for congenital hearing impairment but otherwise had no past medical history of note, and was not taking any medications. There was no family history of note.

Examination revealed the presence of a healthy athletic 14-year-old female. Her heart rate was 58bpm and regular, and her blood pressure 108/78 mmHg. Cardiovascular examination revealed a JVP of 3cm and was otherwise unremarkable with normal heart sounds. Gastrointestinal examination and neurological examinations were likewise unremarkable.

Initial investigations revealed the following results:

ECG: 57bpm normal, sinus rhythm QRS 112 ms, PR 122ms, QTc 502ms, normal ST and T wave morphology.

What is the most likely diagnosis?

○ Brugada syndrome ×
○ Romano Ward syndrome ×
○ Wolff Parkinson White syndrome ×
○ Jervell & Lange-Nielsen syndrome ×
○ Hypertrophic obstructive cardiomyopathy ×

Submit answer

Reference ranges

A 14-year-old female presented to her GP having collapsed at school. Her friends stated that whilst she was playing hockey she inexplicably collapsed and had lost consciousness for a few seconds. This was her first episode of collapse. No seizure activity was observed, and she made a full and spontaneous recovery a few moments later. She denied the presence of prodromal symptoms, and other than occasional palpitations upon exertion she was otherwise well. She had a cochlear implant inserted in early childhood for congenital hearing impairment but otherwise had no past medical history of note, and was not taking any medications. There was no family history of note.

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Initial investigations revealed the following results:

ECG: 57bpm normal, sinus rhythm QRS 112 ms, PR 122ms, QTc 502ms, normal ST and T wave morphology.

What is the most likely diagnosis?

Brugada syndrome	3%
Romano Ward syndrome	26%
Wolff Parkinson White syndrome	2%
Jervell & Lange-Nielsen syndrome	66%
Hypertrophic obstructive cardiomyopathy	4%

Inherited long QT syndrome, sensorineural deafness - Jervell and Lange-Nielsen syndrome

Important for me Less important

This female has long QT syndrome (LQTS), as per the findings of her ECG in combination with a history of collapse on exertion. The main differential diagnosis here is Romano-Ward syndrome, which is also a LQTS but is not associated with deafness, as opposed to Jervell & Lange-Nielsen syndrome, which is associated with deafness. A normal cardiac examination with no evidence of hypertrophy on ECG would make the diagnosis of hypertrophic obstructive cardiomyopathy less likely.

👍 👎 Discuss (4) Improve

Next question >

Long QT syndrome ★

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
- Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) - Romano-Ward syndrome (no deafness)	- amiodarone, sotalol, class 1a antiarrhythmic drugs - tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) - methadone - chloroquine - terfenadine** - erythromycin - haloperidol - ondanestron	- electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia - acute myocardial infarction - myocarditis - hypothermia - subarachnoid haemorrhage

Features

- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management

- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

2010 Drug-Induced Long QT syndrome

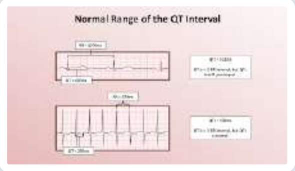
Life in the Fast Lane 👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link

Report broken link

Media



Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **16.7%**

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Question 61 of 129

A 59-year-old woman presents to the emergency department with shortness of breath. She has been progressively getting worse over two weeks and has now started to feel short of breath on rest. She is known to have mitral valve prolapse and is awaiting surgery but has so far not been given a date. She also has polycystic ovarian syndrome, type 2 diabetes and depression. She takes metformin, sertraline and furosemide.

On examination, she looks unwell. She has bilateral crepitations with no wheeze on auscultation of her chest, a raised JVP, and a systolic murmur. A chest X-ray shows pulmonary oedema. She is treated with IV diuretics but remains breathless and hypoxic. What kind of ventilatory support would be most appropriate?

○ No ventilator support is appropriate ×

○ Negative pressure ventilation ×

○ Intubation ×

○ Bilevel positive airway pressure (BIPAP) ×

○ Continuous positive airway pressure (CPAP) ×

Submit answer

Reference ranges

Score: **16.7%**

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A 59-year-old woman presents to the emergency department with shortness of breath. She has been progressively getting worse over two weeks and has now started to feel short of breath on rest. She is known to have mitral valve prolapse and is awaiting surgery but has so far not been given a date. She also has polycystic ovarian syndrome, type 2 diabetes and depression. She takes metformin, sertraline and furosemide.

On examination, she looks unwell. She has bilateral crepitations with no wheeze on auscultation of her chest, a raised JVP, and a systolic murmur. A chest X-ray shows pulmonary oedema. She is treated with IV diuretics but remains breathless and hypoxic. What kind of ventilatory support would be most appropriate?

No ventilator support is appropriate	2%
Negative pressure ventilation	1%
Intubation	2%
Bilevel positive airway pressure (BIPAP)	14%
Continuous positive airway pressure (CPAP)	80%

Acute heart failure not responding to treatment - consider CPAP

Important for me Less important

The correct answer is continuous positive airway pressure (CPAP). This is a patient with a known mitral valve prolapse presenting with decompensation. It can be assumed that the lesion is significant as she is due to have surgery to repair it. She has developed signs of pulmonary oedema and she is not responding to treatment with diuretics. Vasodilators and opiates could be a plan of further medical management, but CPAP could help reduce pulmonary oedema. BIPAP would be appropriate in ventilatory failure in COPD, whilst negative pressure ventilation is more of historical treatment for polio.

Mitral valve prolapse has a prevalence of 2-3% in the population and the majority of cases will be asymptomatic. Risk factors for progression the severe disease include:

- Comorbidities: atrial fibrillation, left sided cardiac failure, hypertension and obesity.
- Echocardiogram findings: mitral leaflet thickness >5mm, prolapse of the posterior leaflet, moderate or severe regurgitation.
- Stress echocardiogram findings: regurgitation during exercise but not at rest.

👍 👎 💬 Discuss (3) Improve

Next question >

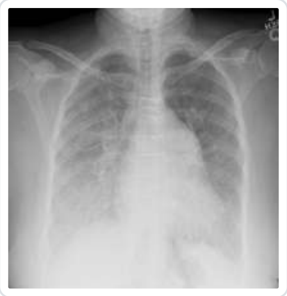
Heart failure: acute management ★

Recommended treatments for all patients

- IV loop diuretics
    - e.g. furosemide or bumetanide

Possible additional treatments

- oxygen
    - this should be given in line with British Thoracic Society guidelines, i.e. keep oxygen saturations at 94-98%
- vasodilators
    - nitrates should not be routinely given to all patients
    - they may, however, have a role if concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease
    - the major side-effect/contraindication is hypotension



Patients with respiratory failure

- CPAP

Patients with hypotension (e.g. < 85 mmHg)/cardiogenic shock

- this can be a difficult scenario to manage
    - some of the treatments typically used for acute heart failure (e.g. loop diuretics and nitrates) may exacerbate the hypotension
- inotropic agents
    - e.g. dobutamine
    - should be considered for patients with severe left ventricular dysfunction who have potentially reversible cardiogenic shock
- vasopressor agents
    - e.g. norepinephrine
    - normally only used if insufficient response to inotropes and evidence of end-organ hypoperfusion
- mechanical circulatory assistance
    - e.g. intra-aortic balloon counterpulsation or ventricular assist devices

Other treatments

- opiates
    - NICE state 'do not routinely offer opiates to people with acute heart failure'
    - they were previously used routinely and help to reduce dyspnoea/distress in patients
    - however some studies have suggested increased morbidity in patients given opiates

General points

- regular medication for heart failure such as beta-blockers ACE-inhibitors should be continued
    - beta-blockers should only be stopped if the patient has heart rate less than 50 beats per minute, second or third degree

atrioventricular block, or shock

123

Next question

Textbooks

High-yield textbook

Extended textbook

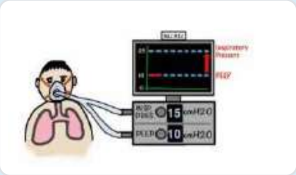
Links

NICE 👍 6 👎 9

2014 Acute heart failure: diagnosis and management

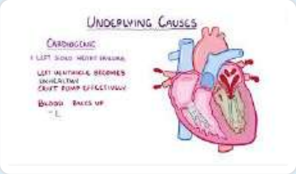
Suggest link Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 👍 17 👎 0


UNDERLYING CAUSES
CARDIOGENIC

Pulmonary oedema

Osmosis - YouTube 👍 4 👎 0

Report broken media

Score: **18%**

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Question 62 of 129

Whilst covering medical receiving overnight, you are referred a 52-year-old man presenting to the Emergency Department with palpitations. He describes 'fluttering in the chest' for the last 48 hours. His past medical history is remarkable only for type 1 diabetes, adequately controlled with insulin. His admission ECG shows atrial fibrillation with a ventricular rate of 130-140 beats per minute. Examination reveals; blood pressure 88/60 mmHg, with capillary refill 3 seconds. His chest is clear to auscultation. He scores 15/15 on the Glasgow coma score. The patient feels light headed but is otherwise uncomplaining.

What is the most appropriate next step in management?

- ○ Monitor and observe the patient on the Coronary Care Unit ×
- ○ Load with 300mg, intravenous amiodarone ×
- ○ Attempt vagal manoeuvres ×
- ○ Give 5mg intravenous metoprolol as a slow bolus ×
- ○ Arrange DC cardioversion ×

Submit answer

Reference ranges

Score: **18%**

1 ✗
2 ✓
3 ✗

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What is the most appropriate next step in management?

Monitor and observe the patient on the Coronary Care Unit	1%
Load with 300mg, intravenous amiodarone	6%
Attempt vagal manoeuvres	3%
Give 5mg intravenous metoprolol as a slow bolus	5%
Arrange DC cardioversion	85%

In the context of a tachyarrhythmia, a systolic BP < 90 mmHg → DC cardioversion

Important for me Less important

The Resuscitation Council (UK) gives clear advice on the management of tachyarrhythmias (see link below for the relevant algorithm). Essentially patients with tachyarrhythmias should be categorised first by the presence or absence of adverse features, these are:

- 1) Shock - Systolic blood pressure of less than 90mmHg, cold extremities, prolonged capillary refill time etc.
- 2) Syncope
- 3) Heart failure pulmonary oedema plus or minus raised jugular venous pressure

- 4) Myocardial ischaemia usually represented by chest pain or typical ECG changes
- 5) Extreme rate very fast rates may also be considered an adverse feature (150 beats per minute is suggested by the Resus Council guideline)

Patients with one or more of the above features are recommended to undergo electrical cardioversion in the first instance.
Patients who do not have any adverse features are then sub stratified as having either a narrow or broad complex tachycardia and then into regular or irregular rhythms (see link below for further details of management).

In the scenario given above, the patient did demonstrate adverse features in the form of shock (systolic pressure < 90mmHg and prolonged capillary refill time) and as such electrical cardioversion is the best option. Note that the Resus Council guideline suggests correcting reversible causes such as electrolyte abnormalities in the first instance, however, this was not one of the options given.

👍 | 👎 | Discuss (8) | Improve

Next question >

Peri-arrest rhythms: tachycardia ★

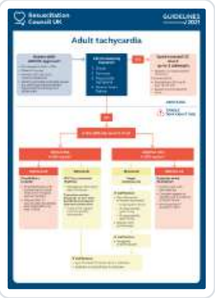
The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:
- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia

- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given. Up to 3 shocks can be given; after this expert help should be sought.

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment guidelines can be found at the Resuscitation Council website, below is a very limited summary:


Resuscitation Council UK
GUIDELINES 2021
Adult tachycardia

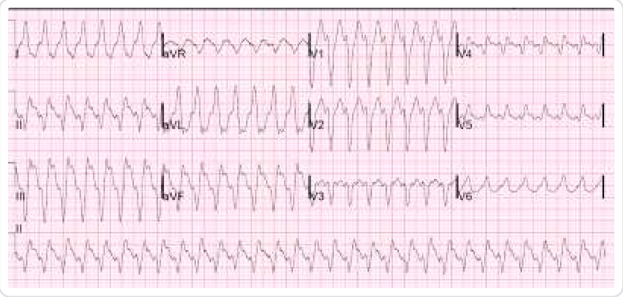
Broad-complex tachycardia

Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion

Irregular

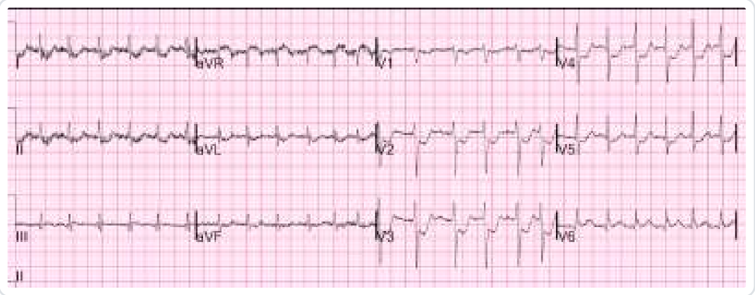
- seek expert help. Possibilities include:
    - atrial fibrillation with bundle branch block - the most likely cause in a stable patient
    - atrial fibrillation with ventricular pre-excitation
    - torsade de pointes


aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Narrow-complex tachycardia

Regular
- vagal manoeuvres followed by IV adenosine
- if the above is unsuccessful consider a diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular
- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control: beta-blockers are usually first-line unless there is a contraindication



123

Next question

Textbooks

High-yield textbook

Extended textbook

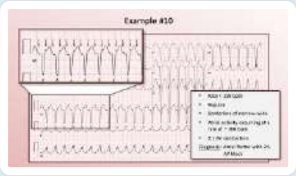
Links

Resuscitation Council UK 👍 30 👎 9

2021 Tachycardia algorithm

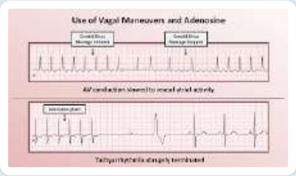
Suggest link Report broken link

Media



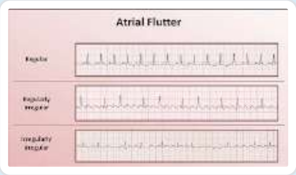
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 5 👎 0


Use of Vagal Maneuvers and Adenosine

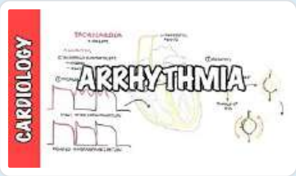
Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 4 👎 0


Atrial Flutter

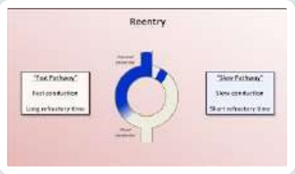
Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 1


CARDIOLOGY
ARRHYTHMIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 1


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube

👍 2 👎 1

Report broken media

Score: **19.4%**

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A 45-year-old man presents to the Emergency Department with shortness of breath, heavy chest pain and syncope. The chest pain and shortness of breath have been worsening over the last week. He is currently undergoing adjunct chemotherapy for a non-resectable soft tissue sarcoma. He has known metastasis in his thorax and mediastinum.

Hb	90 g/L	Male: (135-180) Female: (115 - 160)
Platelets	100 * 10^9/L	(150 - 400)
WBC	12.3 * 10^9/L	(4.0 - 11.0)

Na^+	131 mmol/L	(135 - 145)
K^+	3.1 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	145 μmol/L	(55 - 120)

On physical examination his JVP is raised at 6cm, there are no precordial thrills, and he has quiet S1 and S2.

Observations show:
- Heart rate of 115/min and regular
- Blood pressure 95/55mmHg
- Respiratory rate 28/min
- Temperature 36.5°C
- AVPU - A

What is the more specific ECG finding associated with this diagnosis?

○ Electrical alternans ×

○ RBBB ×

○ S1Q3T3 ×

○ ST elevation in leads V1-4 ×

○ Sinus tachycardia ×

Submit answer

Reference ranges ⌵

Score: **19.4%**

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Question 63 of 129 ✓

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Observations show:
- Heart rate of 115/min and regular
- Blood pressure 95/55mmHg
- Respiratory rate 28/min
- Temperature 36.5°C
- AVPU - A

What is the more specific ECG finding associated with this diagnosis?

Electrical alternans	70%
RBBB	3%

S1Q3T3	17%
ST elevation in leads V1-4	3%
Sinus tachycardia	8%

Electrical alternans is suggestive of cardiac tamponade

Important for me Less important

Electrical alternans is the correct answer. The patient has metastatic disease within the thorax and mediastinum increasing his risk of a pericardial effusion. Electrical alternans is seen in massive pericardial effusions as an alternating height of QRS complex across the rhythm strip. Other ECG findings to look for are tachycardia and low voltage QRS. The website Life in the Fast Lane has a library of ECGs with examples of this.

RBBB is the incorrect answer because this is not a specific finding for massive pericardial effusion. There are a variety of causes of RBBB. Examples include congenital heart disease, cardiomyopathy, pulmonary embolus, right ventricular hypertrophy, ischaemic heart disease and others. In an acute PE setting, which is a differential, in this case, you would also look for right heart strain due to the sudden increase in right ventricular pressure caused by the obstruction to the pulmonary vasculature.

S1Q3T3 is the incorrect answer as this is associated with a pulmonary embolism (PE). However, this is only seen in approximately 15% of PEs. The more common ECG finding is sinus tachycardia.

ST elevation in leads V1-4 is the incorrect answer because this is seen in an anterior occlusive, or type 1, myocardial infarction. This typically affects the left anterior descending artery and confers a high risk of a ventricular arrhythmia. The patient requires urgent PCI or, if that is not available within 120 minutes, then thrombolysis.

Sinus tachycardia is the incorrect answer as it is a non-specific finding on ECG. You would expect a patient with a massive pericardial effusion to mount a tachycardic response. This is because a decrease in diastolic relaxation and the subsequent volume of ventricular filling decreases your stroke volume. To maintain an adequate cardiac output, the patient will increase HR. As CO = SV x HR. However, this is a non-specific finding and is seen in all types of shock; septic, cardiogenic, distributive and hypovolaemic. Answer 1 is a much more specific ECG finding.

👍 👎 Discuss (6) Improve

Next question >

Cardiac tamponade ★

Cardiac tamponade is characterized by the accumulation of pericardial fluid under pressure.

Classical features - Beck's triad:
- hypotension
- raised JVP
- muffled heart sounds

Other features:
- dyspnoea
- tachycardia
- an absent Y descent on the JVP - this is due to the limited right ventricular filling
- pulsus paradoxus - an abnormally large drop in BP during inspiration
- Kussmaul's sign - much debate about this
- ECG: electrical alternans

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

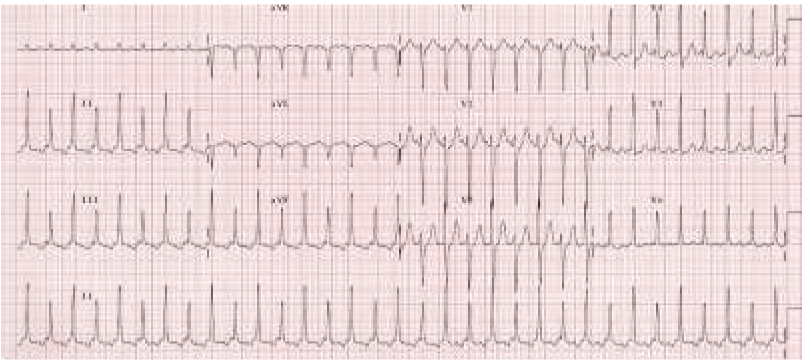
	Cardiac tamponade	**Constrictive pericarditis**
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present

	Cardiac tamponade	**Constrictive pericarditis**
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Management

- urgent pericardiocentesis



© Image used on license from Dr Smith, University of Minnesota

An ECG demonstrating electrical alternans. Note the alternation of QRS complex amplitude between beats.

+ Q 123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 👍 12 👎 4

ECG Findings in Massive Pericardial Effusion

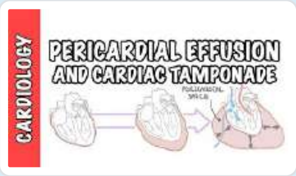
Suggest link

Report broken link

Media

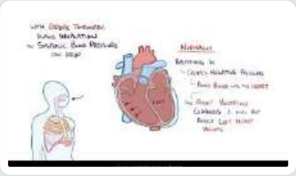
Echocardiogram of a Patient With Cardiac Tamponade

JAMA - YouTube 👍 12 👎 0


CARDIOLOGY
PERICARDIAL EFFUSION
AND CARDIAC TAMPONADE

Cardiac tamponade

Armando Hasudungan - YouTube 👍 1 👎 0



Cardiac tamponade

Osmosis - YouTube 👍 0 👎 0

Report broken media

Score: **20.6%**

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A 24-year-old man is brought in by ambulance to the emergency department after a witnessed collapse. He was unconscious for 10-15 seconds before quickly recovering. There were no preceding symptoms and no witnessed seizure activity. He has no past medical history and is not on any regular medications.

Observations:

- Heart rate 82 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 16/minute
- Oxygen saturations 96% on room air
- Temperature 37C

The examination is unremarkable and his Glasgow coma scale is 15/15.

An ECG demonstrates possible coved ST elevation in leads V1-V3 without reciprocal changes.

Troponin	6 ng/L	(< 14)

Given the likely diagnosis, the administration of which anti-arrhythmic will make the ECG changes more apparent?

○ Dobutamine

○ Flecainide

○ Isoprenaline

○ Metoprolol

○ Quinidine

Submit answer

Question 64 of 129 ✗

A 24-year-old man is brought in by ambulance to the emergency department after a witnessed collapse. He was unconscious for 10-15 seconds before quickly recovering. There were no preceding symptoms and no witnessed seizure activity. He has no past medical history and is not on any regular medications.

Observations:

- Heart rate 82 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 16/minute
- Oxygen saturations 96% on room air
- Temperature 37C

The examination is unremarkable and his Glasgow coma scale is 15/15.

An ECG demonstrates possible coved ST elevation in leads V1-V3 without reciprocal changes.

Troponin	6 ng/L	(< 14)

Given the likely diagnosis, the administration of which anti-arrhythmic will make the ECG changes more apparent?

Dobutamine	7%
Flecainide	75%
Isoprenaline	6%
Metoprolol	6%
Quinidine	6%

ECG changes in Brugada syndrome are more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases

Important for me Less important

Flecainide is the correct answer. The patient presents with syncope and ECG changes that raise the possibility of Brugada syndrome (coved ST elevation in leads V1-V3). Administration of flecainide can make these ECG changes more apparent and aid diagnosis.

Dobutamine is incorrect. This medication can be used in stress echocardiography to determine if there is any inducible ischaemia but is not used to make the ECG changes of Brugada syndrome more apparent.

Metoprolol is incorrect. Typically, a short-acting beta-blocker is administered prior to CT coronary angiography with the aim of reducing the heart rate to a level of approximately 60 to facilitate interpretation of this form of imaging. It is not used to unmask the ECG changes of Brugada syndrome.

Quinidine is incorrect. This has been suggested in some reports to be useful as an anti-arrhythmic in Brugada syndrome. It does not make the ECG changes more prominent.

Isoprenaline is incorrect. This has been shown to normalise the electrocardiographic pattern in Brugada syndrome.

Discuss (2) Improve

Next question

Brugada syndrome

Brugada syndrome is a form of inherited cardiovascular disease with may present with sudden cardiac death. It is inherited in an autosomal dominant fashion and has an estimated prevalence of 1:5,000-10,000. Brugada syndrome is more common in Asians.

Pathophysiology

- a large number of variants exist

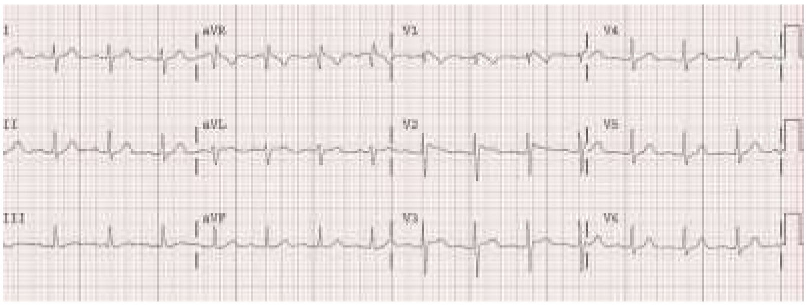
- around 20-40% of cases are caused by a mutation in the SCN5A gene which encodes the myocardial sodium ion channel protein

ECG changes

- convex ST segment elevation > 2mm in > 1 of V1-V3 followed by a negative T wave
- partial right bundle branch block
- the ECG changes may be more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases of Brugada syndrome

Management

- implantable cardioverter-defibrillator



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ECG showing Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

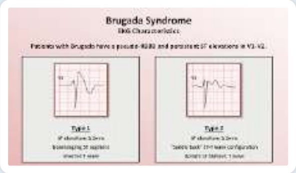
Links

Life in the fast lane 👍 29 👎 5

Brugada syndrome

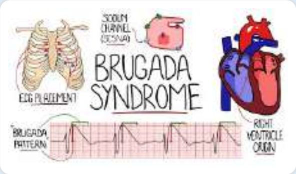
Suggest link Report broken link

Media


Brugada Syndrome
EKG Characteristics
Type 1
Type 2

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 12 👎 0


SODIUM CHANNEL (SCN5A)
BRUGADA SYNDROME
ECG PLACEMENT
BRUGADA PATTERN
RIGHT VENTRICLE ORIGIN

Brugada syndrome

Rhesus Medicine - YouTube 👍 1 👎 0

Report broken media

Score: **20.3%**

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59	✗
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64	✗

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Back to top

A 25-year-old male is brought to the emergency department with an episode of syncope, which occurred without warning. He has no past medical history and does not take any regular medications. There is no history of substance misuse.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/88 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

The examination is unremarkable.

An ECG demonstrates T wave inversion in leads V1-V3.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.8 * 10^9/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin	4 ng/L	(<14)

An echocardiogram demonstrates a dilated, hypokinetic right ventricle.

A CT pulmonary angiogram excludes a pulmonary embolism.

Given the likely diagnosis, what is the most specific ECG finding suggestive of this condition?

- ○ Coved ST segment elevation in V1-V2 ×
- ○ Delta wave ×
- ○ Epsilon wave ×
- ○ PR depression ×
- ○ Low voltage QRS and electrical alternans ×

Submit answer

Reference ranges ⌄

Score: **20.3%**

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Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin	4 ng/L	(<14)

An echocardiogram demonstrates a dilated, hypokinetic right ventricle.

A CT pulmonary angiogram excludes a pulmonary embolism.

Given the likely diagnosis, what is the most specific ECG finding suggestive of this condition?

Coved ST segment elevation in V1-V2	14%
Delta wave	4%
Epsilon wave	76%
PR depression	3%
Low voltage QRS and electrical alternans	3%

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

Important for me Less important

Epsilon wave is correct. The patient presents with syncope without warning, suggesting a cardiogenic cause. T wave inversion in leads V1-V3 and an echocardiogram demonstrating a dilated, hypokinetic right ventricle is characteristic of arrhythmogenic right ventricular dysplasia (ARVD). The most specific finding for this condition is an epsilon wave, which is a small positive deflection at the end of the QRS complex).

Delta wave is incorrect. This is a slurred upstroke of the QRS complex and suggests Wolff-Parkinson-White (WPW) syndrome.

Coved ST segment elevation in V1-V2 is incorrect. This is characteristic of Brugada syndrome, which is another cause of sudden cardiac death.

PR depression is incorrect. The ECG finding, in the correct clinical context, is associated most specifically with pericarditis.

Low voltage QRS and electrical alternans is incorrect. This combination of ECG findings suggests a massive pericardial effusion.

👍 👎 Discuss Improve

Next question ›

Arrhythmogenic right ventricular cardiomyopathy

★

Arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic right ventricular dysplasia or ARVD) is a form of inherited cardiovascular disease which may present with syncope or sudden cardiac death. It is generally regarded as the second most common cause of sudden cardiac death in the young after hypertrophic cardiomyopathy.

Pathophysiology

- inherited in an autosomal dominant pattern with variable expression
- the right ventricular myocardium is replaced by fatty and fibrofatty tissue
- around 50% of patients have a mutation of one of the several genes which encode components of desmosome

Presentation

- palpitations
- syncope
- sudden cardiac death

Investigation

- ECG abnormalities in V1-3, typically T wave inversion. An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex
- echo changes are often subtle in the early stages but may show an enlarged, hypokinetic right ventricle with a thin free wall
- magnetic resonance imaging is useful to show fibrofatty tissue

Management

- drugs: sotalol is the most widely used antiarrhythmic
- catheter ablation to prevent ventricular tachycardia
- implantable cardioverter-defibrillator

Naxos disease

- an autosomal recessive variant of ARVC
- a triad of ARVC, palmoplantar keratosis, and woolly hair

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 👍 13 👎 7

Arrhythmogenic right ventricular cardiomyopathy ECG

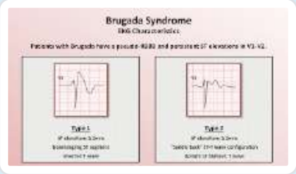
Suggest link Report broken link

Media

CARDIOLOGY
Arrhythmogenic Right Ventricular Cardiomyopathy
(ARVC)

Arrhythmogenic right ventricular cardiomyopathy

Armando Hasudungan - YouTube 👍 8 👎 0


Brugada Syndrome
Type 1
Type 2

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 11 👎 2

Report broken media

Score: **20%**

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Question 66 of 129

A 78-year-old man who is usually fit and well and takes levothyroxine for hypothyroidism presents to the acute medical take after two episodes of syncope. He recalls sitting at the dinner table and passing out with no dizziness prior. He can usually walk 100 metres at a time.

On examination, he has a loud systolic murmur heard over the 2nd intercostal space on the left side radiating to the carotid. He has a slow rising pulse and his chest is clear. No JVP is seen and there is no ankle oedema.

Chest x-ray	enlarged heart with calcification of the aortic knuckle
ECG	sinus, 75 beats per minute
ECHO	Aortic valve cross sectional area of 0.8mm² with a pressure gradient of 42mmHg, cusps appear calcified and poorly mobile

What other investigation should be performed?

- ○ Coronary angiography ×
- ○ Thyroid function test ×
- ○ CT chest ×
- ○ Treadmill ECG testing ×
- ○ HbA1c ×

Submit answer

Reference ranges

Score: **20%**

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Chest x-ray	enlarged heart with calcification of the aortic knuckle
ECG	sinus, 75 beats per minute
ECHO	Aortic valve cross sectional area of $0.8mm^2$ with a pressure gradient of 42mmHg, cusps appear calcified and poorly mobile

What other investigation should be performed?

Coronary angiography	81%
Thyroid function test	6%
CT chest	5%
Treadmill ECG testing	7%
HbA1c	1%

Aortic stenosis co-occurs with atherosclerotic disease -> perform angiogram prior to surgical intervention

Important for me Less important

This gentleman meets the ECHO criteria for severe aortic stenosis and should be listed for aortic valve replacement (AVR) immediately. He shows no signs of failure or haemodynamic compromise and is therefore fit. Given that AVR involves opening the chest, it is a perfect time to perform coronary artery bypass grafting and since there is a correlation of aortic stenosis with atherosclerosis, it is beneficial to perform an angiogram prior to the AVR procedure. The patient effectively gets two procedures for one. CT chest would be important is he had respiratory disease. HbA1c and thyroid function tests are not essential unless there was a suspicion that these were abnormal. Treadmill testing is contraindicated in symptomatic and severe aortic stenosis where is can cause syncope.

👍 | 👎 | Discuss (3) | Improve

Next question >

Aortic stenosis ★

Clinical features of symptomatic disease

- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur
    - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
    - classically radiates to the carotids
    - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)

- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management
- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
  - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
  - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
  - may be used in children with no aortic valve calcification
  - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

Guidelines on the management of valvular heart disease

Suggest link Report broken link

Media

Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1

AORTIC VALVE DISEASE
Osmosis.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2

AORTIC STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

Report broken media

Score: **19.7%**

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A 25-year-old male presents to the emergency department with palpitations. His observations are performed, heart rate 180/min, blood pressure 134/78 mmHg, respiratory rate 16/min. His past medical history includes asthma for which he takes salbutamol PRN and fluticasone BD. Examination reveals a clear chest. Heard sounds are too rapid to distinguish S1 and S2. ECG reveals a narrow complex tachycardia, consistent with supraventricular tachycardia (SVT). Multiple attempts at vagal manoeuvres have failed to cardiovert the patient. He remains symptomatic.

Bloods are taken:

Hb	135 g/l
Platelets	344 * 10^9/l
WBC	10.1 * 10^9/l

Na^+	141 mmol/l
K^+	4.2 mmol/l
Creatinine	65 µmol/l
CRP	2 mg/l

Arterial blood gases on room air:

pO2	12.1 kPa
PCO2	4.1 kPa
pH	7.39
Base excess	-0.2

Chest X-ray: No abnormality detected

Which pharmacological agent would be most appropriate for cardioverting this man?

○ Verapamil

○ Adenosine

○ Digoxin

○ Amiodarone

○ Bisoprolol

Submit answer

Reference ranges

Score: **19.7%**

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Chest X-ray: No abnormality detected

Which pharmacological agent would be most appropriate for cardioverting this man?

Verapamil	71%
Adenosine	21%
Digoxin	1%
Amiodarone	7%
Bisoprolol	1%

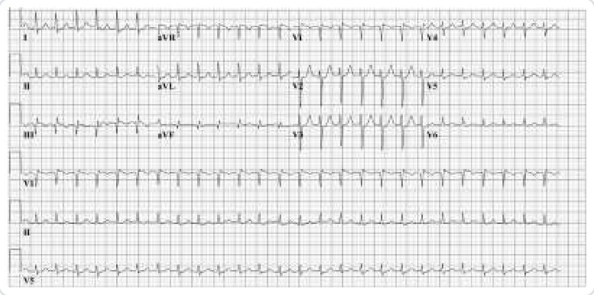
Use of adenosine is contraindicated in patients with asthma as it can cause bronchospasm. The next best option here is verapamil, which slows atrioventricular (AV) nodal conduction. The other options are not widely used to restore sinus rhythm in SVT.

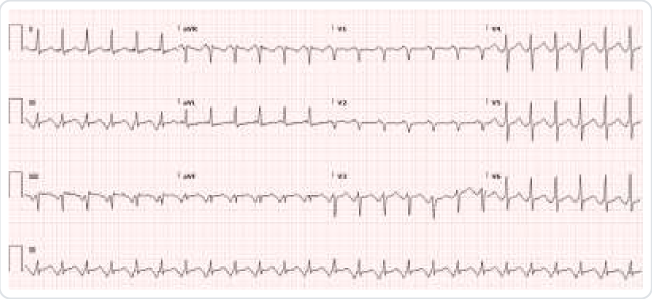
Discuss (2) Improve

Next question

Supraventricular tachycardia

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
    - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
    - carotid sinus massage
- intravenous adenosine
    - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
    - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

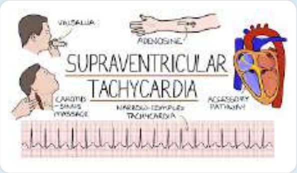
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

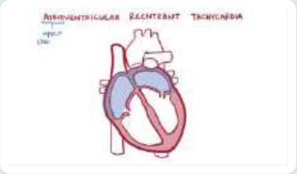
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

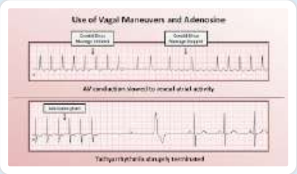
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

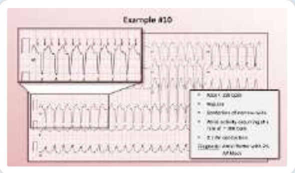
Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0


Use of Vagal Maneuvers and Adenosine

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0



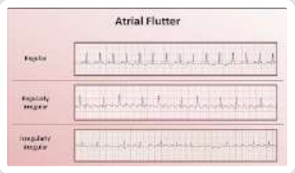
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0

AVRT & AVNRT
Khan Academy

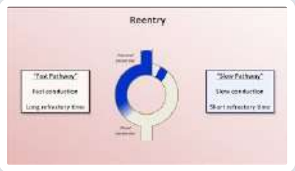
Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0


Atrial Flutter

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0



Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **19.4%**

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Question 68 of 129

A 40-year-old male who is otherwise fit and well presents to clinic with dyspnoea on exertion. This has progressed over the last year to the point where he can only walk 0.5 miles before stopping. He denies having a cough. He has no chest pain. He has smoked 15 cigarettes per day for 25 years. He reports that his father had a myocardial infarction (MI) aged 54 years, but otherwise reports no cardiac-related family history. His body mass index (BMI) is 24 kg/m^2, heart rate 80/min, blood pressure 130/77mmHg, respiratory rate 18/min and he is saturating at 97% on air. Chest auscultation reveals occasional expiratory wheeze and he has no pedal oedema. Auscultation of the heart reveals a fixed split S2. His jugular venous pressure is not elevated.

Chest X-ray: dilated pulmonary vessels.

ECG: sinus rhythm, 78/min, right bundle branch block (RBBB)

What is the likely cause of his exertional dyspnoea?

- ○ Atrial septal defect ×
- ○ Ischaemic heart disease ×
- ○ Bronchial malignancy ×
- ○ Chronic obstructive pulmonary disease ×
- ○ Chronic pulmonary embolism ×

Submit answer

Reference ranges

Score: **19.4%**

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← Question 68 of 129 ✗ ⚐ →

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Chest X-ray: dilated pulmonary vessels.

ECG: sinus rhythm, 78/min, right bundle branch block (RBBB)

What is the likely cause of his exertional dyspnoea?

Atrial septal defect	72%
Ischaemic heart disease	2%
Bronchial malignancy	1%
Chronic obstructive pulmonary disease	13%
Chronic pulmonary embolism	11%

The underlying cause here is likely to be an atrial septal defect. Presentation often occurs late in life with an incidental finding of RBBB. It may not be picked up in infancy as a murmur may not be audible. Presentation with symptoms occurs later in life as the right atrium becomes dilated and hence the cardiac efficiency decreases. A fixed-split S2 is a common finding in such patients.

👍 👎 Discuss (5) Improve

Next question >

Atrial septal defects ★

Atrial septal defects (ASDs) are the most likely congenital heart defect to be found in adulthood. They carry a significant mortality, with 50% of patients being dead at 50 years. Two types of ASDs are recognised, ostium secundum and ostium primum. Ostium secundum are the most common

Features
- ejection systolic murmur, fixed splitting of S2
- embolism may pass from venous system to left side of heart causing a stroke

Ostium secundum (70% of ASDs)
- associated with Holt-Oram syndrome (tri-phalangeal thumbs)
- ECG: RBBB with RAD

Ostium primum
- present earlier than ostium secundum defects
- associated with abnormal AV valves
- ECG: RBBB with LAD, prolonged PR interval

AI + Q 123

Next question >

Textbooks

High-yield textbook

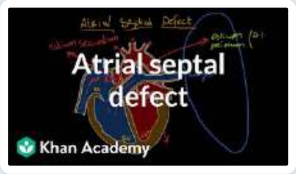
Extended textbook

Media

RA
RV
LV
ASD
VSD
PDA

How to diagnose acyanotic heart diseases (Left to Right shunt) via Cardiac Catheterization.

Medicosis Perfectionalis - YouTube 👍 2 👎 0


Atrial septal defect
Khan Academy

Atrial septal defects

Khan Academy - YouTube 👍 2 👎 1

www.thinklabs.com

Atrial septal defect murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 1 👎 1

ATRIAL SEPTAL DEFECT
Osmosis.org

Atrial septal defect

Osmosis - YouTube 👍 6 👎 4

Report broken media

Score: **19.1%**

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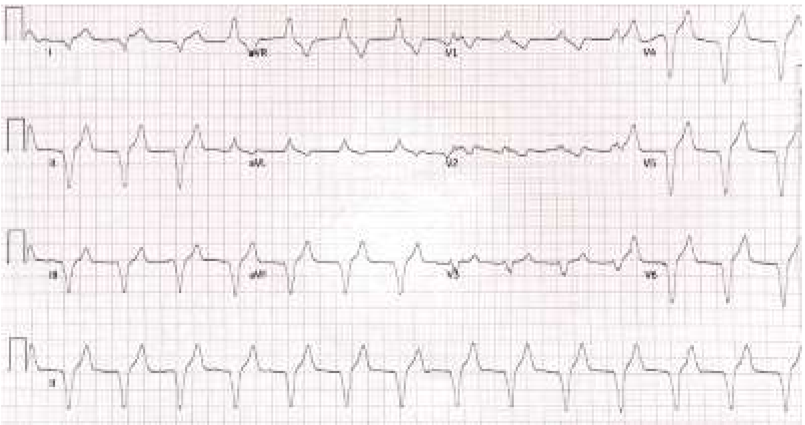
Question 69 of 129

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient was stated on secondary prevention medications and has reports no further issues or symptoms. The patient is currently on aspirin, clopidogrel, bisoprolol, ramipril and atorvastatin.

Examination is normal and vital signs within normal range.

An ECG performed is shown below.



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What definitive management should be advised?

○ Arrange for urgent pacemaker insertion

○ Arrange for urgent repeat PCI

○ No additional management is required

○ Stop the patient's beta blocker and continue all other medications

○ Arrange for urgent 24-hour Holter monitor

Submit answer

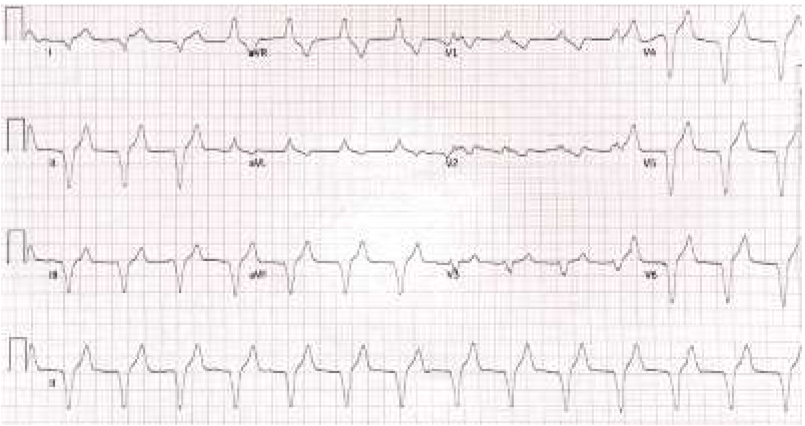
Reference ranges

Question 69 of 129 ✗

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient was stated on secondary prevention medications and has reports no further issues or symptoms. The patient is currently on aspirin, clopidogrel, bisoprolol, ramipril and atorvastatin.

Examination is normal and vital signs within normal range.

An ECG performed is shown below.



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What definitive management should be advised?

Arrange for urgent pacemaker insertion	18%
Arrange for urgent repeat PCI	7%
No additional management is required	64%
Stop the patient's beta blocker and continue all other medications	6%
Arrange for urgent 24-hour Holter monitor	6%

The patient's ECG is a typical example of the benign ventricular ectopic rhythm, accelerated idioventricular rhythm (AIVR). It is associated with atrioventricular dissociation; P waves present but not associated with

QRS complexes, wide QRS complexes (>120ms) and a rate between 50-110 beats per minute. AIVR is common post-MI following the reperfusion of an ischaemic myocardium. It is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable.

Pacemaker insertion is not required as the atrioventricular dissociation and QRS complexes seen in AIVR does not affect the ventricular rate which is normally between 50-110 beats per minute. AIVR is due to a benign ectopic rhythm and so pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Arranging an urgent 24-hour Holter monitor is unnecessary as the patient's ecg is unlikely to change and there is no indication of any other underlying arrhythmia. The patient can be monitored in the outpatient setting as per any other post-MI patient.

👍 👎 Discuss (6) Improve

Next question >

Accelerated idioventricular rhythm

Accelerated idioventricular rhythm (AIVR) is a benign ectopic rhythm of ventricular origin. It usually occurs following the reperfusion of an ischaemic myocardium. It has a rate of 50-110 beats per minute, which helps differentiate it from ventricular bradycardia or ventricular tachycardia.

Pathophysiology:

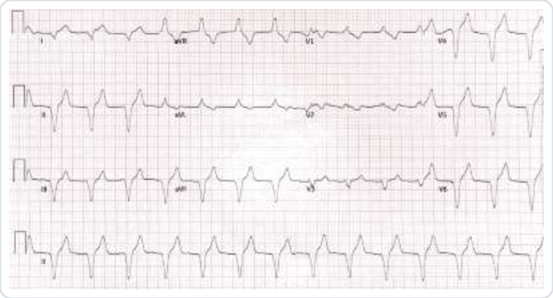
- Reperfusion of ischaemic tissue, electrolyte abnormalities or drug toxicity leads to an increased depolarisation rate of ventricular

myocytes.
- When this depolarisation rate is faster than the rate produced by the sinoatrial node, it becomes the overriding rhythm. Therefore, sinus bradycardia by vagal excess or reduced sympathetic activity can precipitate AIVR.

AIVR has a rate of between 50 -110 beats per minute and, therefore, the patient tends to be haemodynamically stable. However, if the rate is fast or there is extensive damage to the myocardium, the patient can become unstable.

Causes:
- Reperfusion following myocardial infarction - by far the most common cause
- Beta-sympathomimetics (e.g. adrenaline)
- Drug toxicity (digoxin/cocaine)
- Electrolyte imbalance
- Cardiomyopathy, congenital heart disease, myocarditis

AIVR is diagnosed based on electrocardiography (ECG) findings:
- AIVR has a gradual onset and termination as the ventricular rate is only slightly faster than sinus rhythm. This may result in ventricular fusion beats on ECG.
- Atrioventricular dissociation - P waves present but not associated with QRS complexes.
- The ECG will show wide QRS complexes >120ms.
- Rate between 50-110 beats per minute.



Treatment:
- AIVR is usually self-limiting and therefore treatment is not necessary, however, occasionally atropine can be used to increase the sinus rate to overcome AIVR.

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **18.8%**

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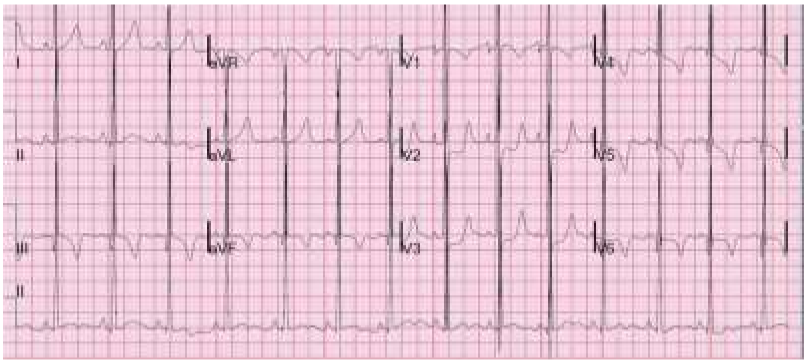
Question 70 of 129

A 32-year-old female presents following a collapse whilst out jogging. The patient reports she experienced a subacute onset of chest discomfort that was quickly followed by symptoms of breathlessness and feeling faint. She thinks she 'blacked out' for a few seconds before recovering. Now she is feeling well with no symptoms.

On further questioning, she does not exercise regularly as she has experienced shortness of breath and chest discomfort in the past. She believes she has had a few episodes of feeling light-headed but has never fainted before. She is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



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What is the patient's most likely diagnosis?

- ○ Brugada syndrome
- ○ Dilated cardiomyopathy
- ○ Hypertrophic obstructive cardiomyopathy
- ○ Wellen's syndrome
- ○ Arrhythmogenic right ventricular dysplasia

Submit answer

Reference ranges ⌄

Score: **18.8%**

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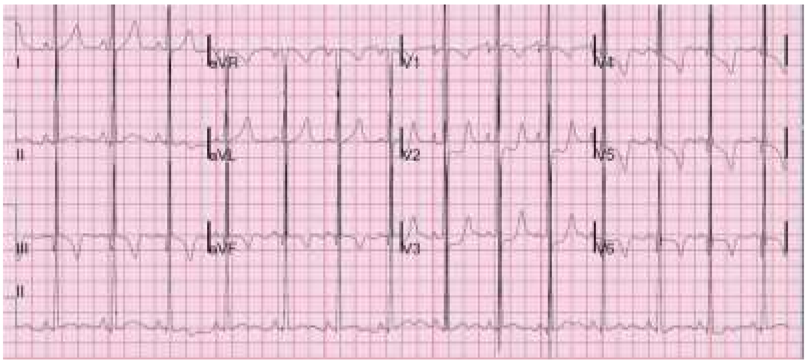

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What is the patient's most likely diagnosis?

Brugada syndrome	10%
Dilated cardiomyopathy	3%
Hypertrophic obstructive cardiomyopathy	67%
Wellen's syndrome	8%
Arrhythmogenic right ventricular dysplasia	13%

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect causing a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncope episodes, commonly during or following exertion. ECG findings, as seen in this case, are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. The diagnosis can be confirmed via cardiac echo and management is dependent on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3, followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and is characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2 and 3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

Arrhythmogenic right ventricular dysplasia (ARVD), is due to a genetic defect affecting the desmosomes of the myocardium. ARVD results in a non-ischemic cardiomyopathy mainly affecting the right ventricle leading to areas of hypokinesia and myocardium replacement with fibrofat. These changes can results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

👍 👎 Discuss (2) Improve

Next question >

Hypertrophic obstructive cardiomyopathy: features ★

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500. HOCM is important as it is the most common cause of sudden cardiac death in the young.

Pathophysiology

- the most common defects involve a mutation in the gene encoding β-myosin heavy chain protein or myosin-binding protein C
- results in predominantly diastolic dysfunction
    - left ventricle hypertrophy → decreased compliance → decreased cardiac output
- characterized by myofibrillar hypertrophy with chaotic and disorganized fashion myocytes ('disarray') and fibrosis on biopsy

Features

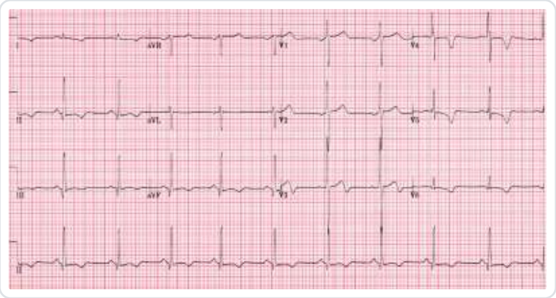
- often asymptomatic
- exertional dyspnoea
- angina
- syncope
    - typically following exercise
    - due to subaortic hypertrophy of the ventricular septum, resulting in functional aortic stenosis
- sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure
- jerky pulse, large 'a' waves, double apex beat
- systolic murmurs
    - ejection systolic murmur: due to left ventricular outflow tract obstruction. Increases with Valsalva manoeuvre and decreases on squatting

  - pansystolic murmur: due to systolic anterior motion of the mitral valve → mitral regurgitation

Associations
- Friedreich's ataxia
- Wolff-Parkinson White

Echo findings - mnemonic - MR SAM ASH
- mitral regurgitation (MR)
- systolic anterior motion (SAM) of the anterior mitral valve leaflet
- asymmetric hypertrophy (ASH)

ECG
- left ventricular hypertrophy
- non-specific ST segment and T-wave abnormalities, progressive T wave inversion may be seen
- deep Q waves
- atrial fibrillation may occasionally be seen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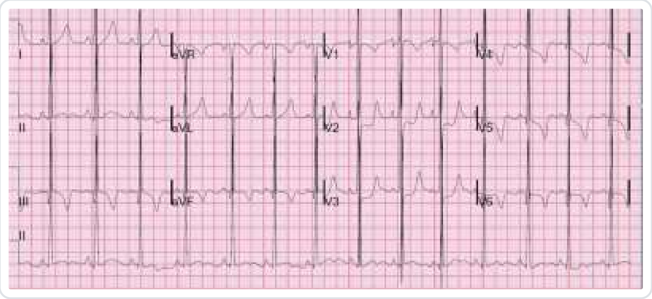
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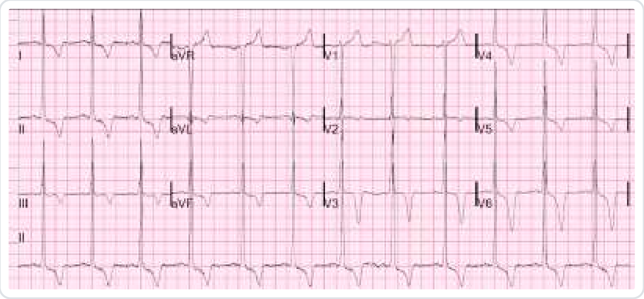
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123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology (ESC) 👍 9 👎 8

2014 ESC HCOM Guidelines

RCP 👍 16 👎 4

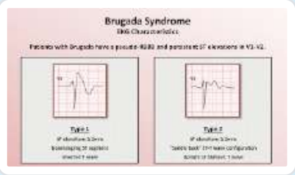
Investigation and treatment of hypertrophic cardiomyopathy

Suggest link Report broken link

Media

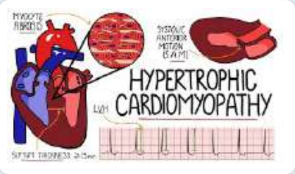
Hypertrophic cardiomyopathy

Osmosis - YouTube 👍 0 👎 0


Brugada Syndrome
Type 1
Type 2

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 6 👎 1


HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic Cardiomyopathy Made Easy

Rhesus Medicine - YouTube 👍 1 👎 1

Hypertrophic Cardiomyopathy heart sounds - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 1 👎 1

CARDIOLOGY
HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic obstructive cardiomyopathy

Osmosis - YouTube 👍 0 👎 4

Report broken media

Score: **18.6%**

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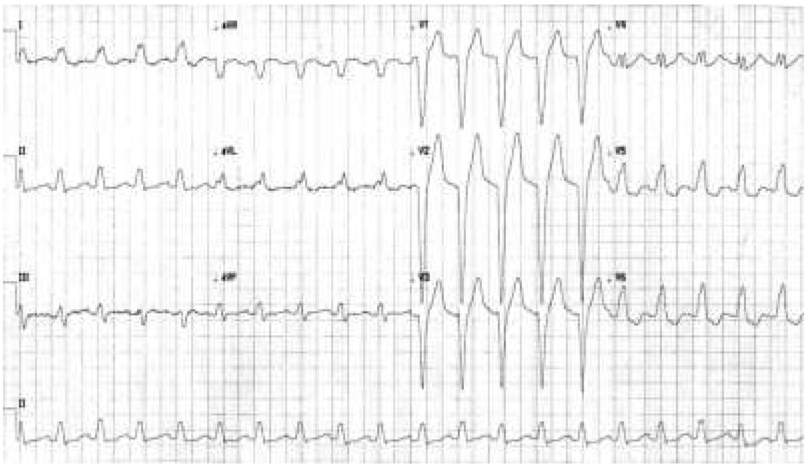
A 72-year-old woman presents with palpitations. Her past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. Her pulse is 120/min, blood pressure 110/76 mmHg and the chest is clear on auscultation.

The ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

What is shown on the ECG?

- ○ Left bundle branch block
- ○ Anterior ST elevation myocardial infarction
- ○ Right bundle branch block
- ○ Narrow complex tachycardia
- ○ Atrial flutter

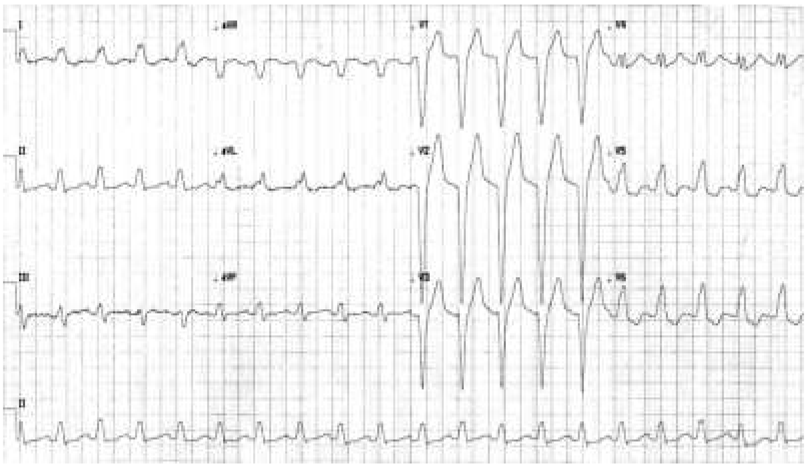
Submit answer

Reference ranges

Question 71 of 129 ✗

A 72-year-old woman presents with palpitations. Her past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. Her pulse is 120/min, blood pressure 110/76 mmHg and the chest is clear on auscultation.

The ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

What is shown on the ECG?

Left bundle branch block	77%
Anterior ST elevation myocardial infarction	10%
Right bundle branch block	7%
Narrow complex tachycardia	3%
Atrial flutter	4%

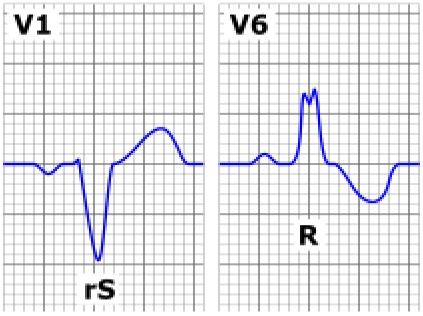
The morphology of the QRS complexes is diagnostic for left bundle branch block (LBBB). This finding can be associated with a wide variety of underlying problems but in an acutely unwell patient myocardial ischaemia needs to be excluded.

Discuss (5) Improve

Next question >

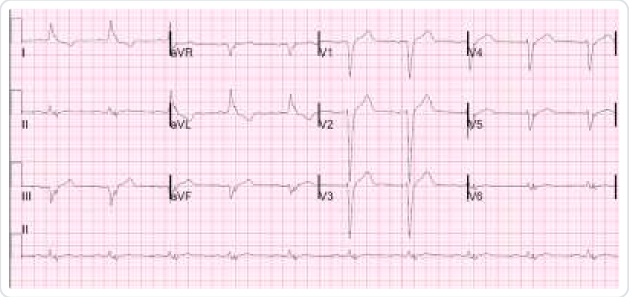
ECG: left bundle branch block

The diagram below shows the typical features of left bundle branch block (LBBB):



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6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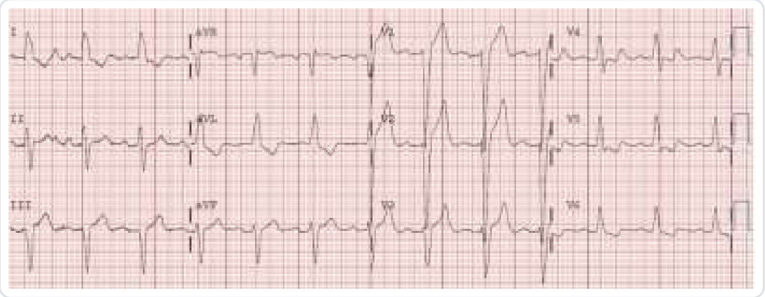
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New LBBB is always pathological. Causes of LBBB include:

- myocardial infarction
    - diagnosing a myocardial infarction for patients with *existing* LBBB is difficult

    - rhe Sgarbossa criteria can help with this - please see the link for more details
- hypertension
- aortic stenosis
- cardiomyopathy
- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

123

Next question >

Textbooks

High-yield textbook

Extended textbook

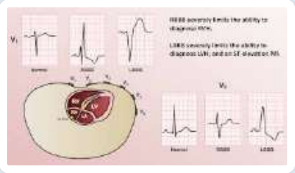
Links

Life in the Fast Lane 👍 33 👎 16

Sgarbossa criteria

Suggest link Report broken link

Media



Intro to EKG Interpretation - Bundle Branch Blocks

Strong Medicine - YouTube 👍 6 👎 2

Bundle branch block

Osmosis - YouTube 👍 0 👎 3

Report broken media

Score: **18.3%**

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71	✗

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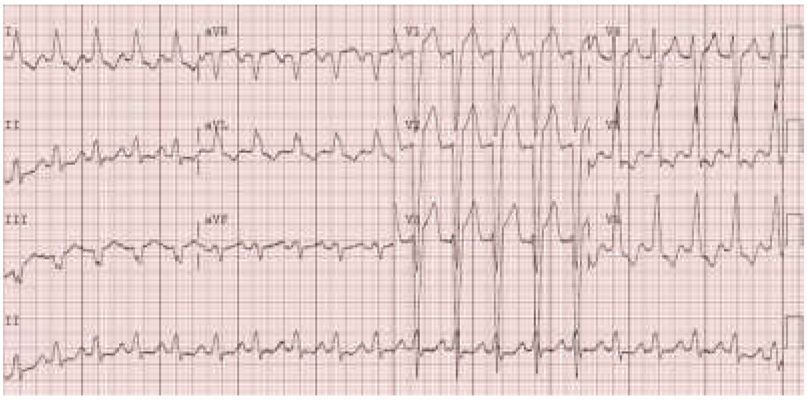
Question 72 of 129

A 58-year-old man presents with intermittent chest pains for the past two days. Whilst you are taking the history he complains of worsening pain and you arrange an ECG immediately:



© Image used on license from Dr Smith, University of Minnesota

He is tachycardic and sweaty with a blood pressure of 128/88mmHg. What does the ECG show?

- ○ Narrow complex tachycardia with ST elevation
- ○ Right bundle branch block with ST elevation
- ○ Left bundle branch block with ST elevation
- ○ Ventricular tachycardia with ST elevation
- ○ Posterior myocardial infarction

Submit answer

Reference ranges

Score: **18.3%**

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72	-

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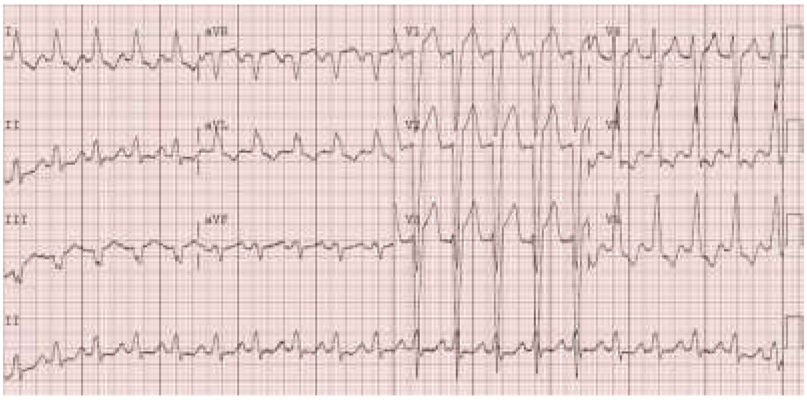


© Image used on license from Dr Smith, University of Minnesota

He is tachycardic and sweaty with a blood pressure of 128/88mmHg. What does the ECG show?

Narrow complex tachycardia with ST elevation	5%
Right bundle branch block with ST elevation	9%
Left bundle branch block with ST elevation	70%
Ventricular tachycardia with ST elevation	12%
Posterior myocardial infarction	4%

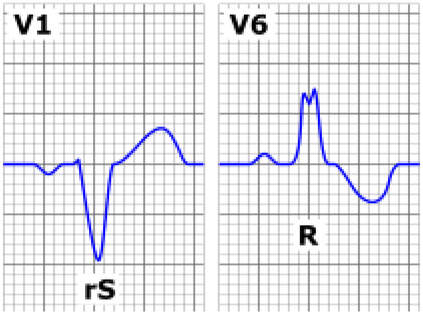
The ECG shows left bundle branch block (LBBB). The relationship between new LBBB and acute coronary syndrome (ACS) is complicated with many cardiologists ignoring the standard guidelines (both UK and US) to initiate reperfusion therapy due to the poor correlation seen in clinical practice between coronary lesions and standard LBBB. The Sgarbossa criteria have therefore been developed to evaluate whether new LBBB represents an ACS. In this particular ECG there is more ST elevation than is normally expected with LBBB indicating a likely ACS.

Discuss (4) | Improve

Next question >

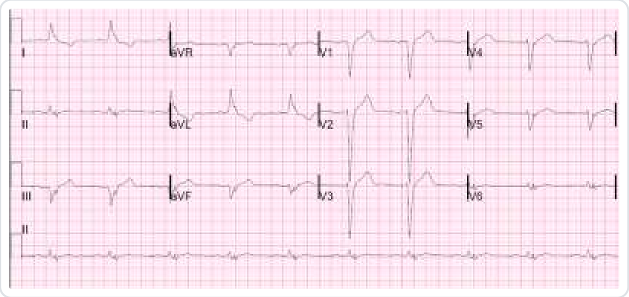
ECG: left bundle branch block

The diagram below shows the typical features of left bundle branch block (LBBB):



One of the most common ways to remember the difference between LBBB and RBBB is WiLLiaM MaRRoW

- in LBBB there is a 'W' in V1 and a 'M' in V6
- in RBBB there is a 'M' in V1 and a 'W' in V6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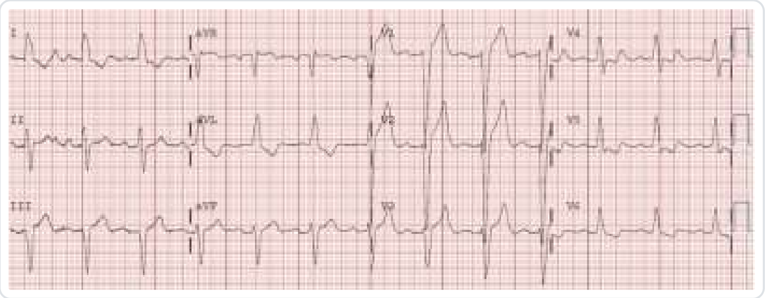
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aVF
V3
V6

New LBBB is always pathological. Causes of LBBB include:

- myocardial infarction
    - diagnosing a myocardial infarction for patients with *existing* LBBB is difficult

    - rhe Sgarbossa criteria can help with this - please see the link for more details
- hypertension
- aortic stenosis
- cardiomyopathy
- rare: idiopathic fibrosis, digoxin toxicity, hyperkalaemia

123

Next question >

Textbooks

High-yield textbook

Extended textbook

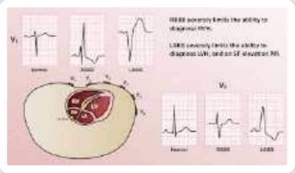
Links

Life in the Fast Lane	33	16

Sgarbossa criteria

Suggest link Report broken link

Media



Intro to EKG Interpretation - Bundle Branch Blocks

Strong Medicine - YouTube 6 2

Bundle branch block

Osmosis - YouTube 👍 0 👎 3

Report broken media

Score: **19.4%**

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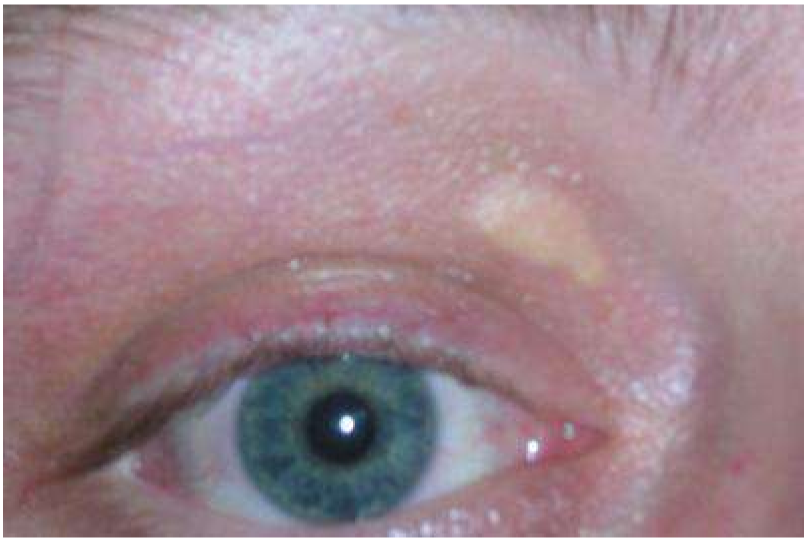
Question 73 of 129

A 40-year-old man presents to surgery as he has noted an abnormality around his right eye:



What is the most likely diagnosis?

○ Hypertriglyceridaemia

○ Hypercholesterolaemia

○ Hypothyroidism

○ Wilson's disease

○ Diabetes mellitus

Submit answer

Reference ranges

Score: **19.4%**

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72 ✓

73 -

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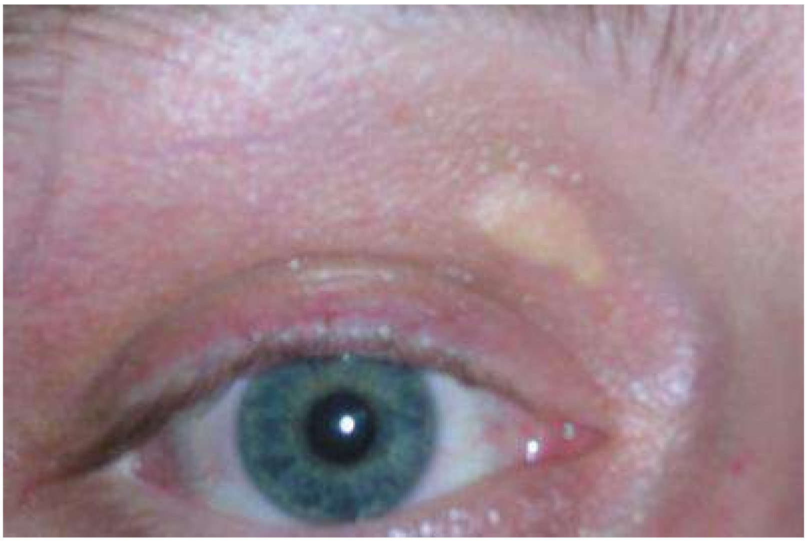
Question 73 of 129 ✗

A 40-year-old man presents to surgery as he has noted an abnormality around his right eye:



What is the most likely diagnosis?

Hypertriglyceridaemia	29%
Hypercholesterolaemia	69%
Hypothyroidism	1%
Wilson's disease	1%
Diabetes mellitus	0%

The correct answer to this question is **Hypercholesterolaemia**. The image shows xanthelasma, which are yellowish plaques that commonly occur on or around the eyelids. Xanthelasma is often associated with hypercholesterolaemia, a condition characterised by high levels of cholesterol in the blood. Hypercholesterolaemia can lead to atherosclerosis and subsequently cardiovascular diseases.

Hypertriglyceridaemia is an incorrect option. While it is true that hypertriglyceridaemia can also cause xanthoma (fatty deposits under the skin), these usually present as eruptive xanthomas (small red-yellow bumps) or tuberous xanthomas (larger nodules), rather than xanthelasma.

Hypothyroidism, another incorrect option, can sometimes be associated with hypercholesterolaemia due to decreased metabolism of lipids. However, hypothyroidism itself would not directly cause xanthelasma. Hypothyroidism presents with symptoms such as fatigue, weight gain, cold intolerance, constipation, and dry skin.

The diagnosis of **Wilson's disease** does not fit the given scenario either. Wilson's disease is a rare inherited disorder causing copper to accumulate in vital organs like the liver and brain. The classic ocular manifestation in Wilson's disease is Kayser-Fleischer rings - copper deposition at the corneal limbus - rather than xanthelasma.

Lastly, **Diabetes mellitus** is also an incorrect answer for this case scenario. Diabetes could lead to various eye problems like diabetic retinopathy or cataracts but it does not cause xanthelasma. It's important for medical students to remember that while diabetes and hyperlipidemias often coexist due to their common risk factors, they are distinct conditions with different manifestations and complications.

👍 | 👎 | Discuss (2) | Improve

Next question >

Hyperlipidaemia: xanthomata ★

Characteristic xanthomata seen in hyperlipidaemia:

Palmar xanthoma
- remnant hyperlipidaemia
- may less commonly be seen in familial hypercholesterolaemia

Eruptive xanthoma are due to high triglyceride levels and present as multiple red/yellow vesicles on the extensor surfaces (e.g. elbows, knees)

Causes of eruptive xanthoma
- familial hypertriglyceridaemia

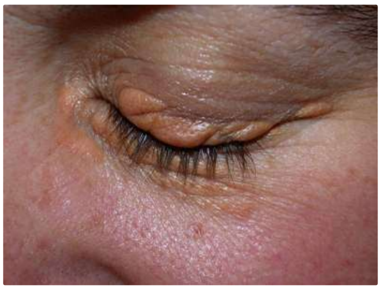
- lipoprotein lipase deficiency

Tendon xanthoma, tuberous xanthoma, xanthelasma
- familial hypercholesterolaemia
- remnant hyperlipidaemia

Xanthelasma

Xanthelasma are yellowish papules and plaques caused by localized accumulation of lipid deposits commonly seen on the eyelid. They are also seen in patients without lipid abnormalities.

Management of xanthelasma, options include:
- surgical excision
- topical trichloroacetic acid
- laser therapy
- electrodesiccation



+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

DermNet NZ 👍 12 👎 6

Xanthomas

Suggest link Report broken link

Score: **19.2%**

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72	✓
73	✗

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Question 74 of 129

A 53-year-old Syrian refugee is referred to the cardiology clinic by her GP with dyspnoea and increasing peripheral oedema. Her past medical history is significant for rheumatic fever as a child. She gives a six month history of increasing dyspnoea on exertion. Her dyspnoea is now so severe that she has marked limitation in activity, being only able to walk 30 meters before stopping because of symptoms. She experiences no chest pain but tells you that she has taken to sleeping in a chair at night and has a chronic cough productive of pink sputum. More recently, she has awoken breathless from sleep terrified that she is going to die. On examination she has clinical evidence of severe mitral stenosis. She has an irregularly-irregular pulse and an ECG confirms atrial fibrillation at a rate of 89 bpm. Auscultation reveals a mid-diastolic murmur, an opening snap and a loud P2. She has bibasal crepitations and pitting oedema to below the knee. Her echo confirms severe mitral stenosis with a mitral valve area of 0.7cm2 and a mean gradient of 12mmHg. There is no evidence of mitral regurgitation and the sonographer comments that she has favourable mitral valve morphology. Assuming a TOE excludes a left apical thrombosis, how should this lady be managed?

○ Diuresis, anticoagulate with one of the oral direct thrombin inhibitors/ factor Xa inhibitors and percutaneous mitral valve balloon valvotomy

○ Diuresis, anticoagulate with warfarin and refer for mitral valve replacement

○ Diuresis, anticoagulate with warfarin and repeat echo in three months time

○ Diuresis, anticoagulate with one of the oral direct thrombin / factor Xa inhibitors and refer for mitral valve replacement

○ Diuresis, anticoagulate with warfarin and percutaneous mitral valve balloon valvotomy

Submit answer

Question 74 of 129 ✓

A 53-year-old Syrian refugee is referred to the cardiology clinic by her GP with dyspnoea and increasing peripheral oedema. Her past medical history is significant for rheumatic fever as a child. She gives a six month history of increasing dyspnoea on exertion. Her dyspnoea is now so severe that she has marked limitation in activity, being only able to walk 30 meters before stopping because of symptoms. She experiences no chest pain but tells you that she has taken to sleeping in a chair at night and has a chronic cough productive of pink sputum. More recently, she has awoken breathless from sleep terrified that she is going to die. On examination she has clinical evidence of severe mitral stenosis. She has an irregularly-irregular pulse and an ECG confirms atrial fibrillation at a rate of 89 bpm. Auscultation reveals a mid-diastolic murmur, an opening snap and a loud P2. She has bibasal crepitations and pitting oedema to below the knee. Her echo confirms severe mitral stenosis with a mitral valve area of 0.7cm2 and a mean gradient of 12mmHg. There is no evidence of mitral regurgitation and the sonographer comments that she has favourable mitral valve morphology. Assuming a TOE excludes a left apical thrombosis, how should this lady be managed?

Diuresis, anticoagulate with one of the oral direct thrombin inhibitors/ factor Xa inhibitors and percutaneous mitral valve balloon valvotomy	13%
Diuresis, anticoagulate with warfarin and refer for mitral valve replacement	37%
Diuresis, anticoagulate with warfarin and repeat echo in three months time	2%
Diuresis, anticoagulate with one of the oral direct thrombin / factor Xa inhibitors and refer for mitral valve replacement	22%
Diuresis, anticoagulate with warfarin and percutaneous mitral valve balloon valvotomy	26%

The correct answer is diuresis, anticoagulate with warfarin and percutaneous mitral valve balloon valvotomy.

The following is taken from the 2014 AHA/ACC consensus guidelines:

Percutaneous mitral balloon valvotomy is recommended for symptomatic patients with severe MS (mitral valve area 1.5 cm2) and favourable valve morphology in the absence of left atrial thrombus or moderate-to-severe MR.

Mitral valve surgery (repair, commissurotomy, or valve replacement) is indicated in severely symptomatic patients (NYHA class III to IV) with severe MS (mitral valve area 1.5 cm2) who are not high risk for surgery and who are not candidates for or who have failed previous percutaneous mitral balloon valvotomy.

Concomitant mitral valve surgery is indicated for patients with severe MS (mitral valve area 1.5 cm2) undergoing cardiac surgery for other indications.

With respect to anti-coagulation, the direct acting/ non-vitamin K antagonists are only indicated in non-valvular AF. Once adequate diuresis has been achieved, the addition of a beta-blocker should also be considered.

Reference

Nishimura et al. 2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary. Circulation. 2014;129: 2440-2492

👍 👎 Discuss (11) Improve

Next question >

Mitral stenosis ★

Mitral stenosis describes the obstruction of blood flow across the mitral valve from the left atrium to the left ventricle. This leads to increases in pressure within the left atrium, pulmonary vasculature and right side of the heart.

It is said that the causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever. Rarer causes that may be seen in the exam include mucopolysaccharidoses, carcinoid and endocardial fibroelastosis

Features
- dyspnoea
  - ↑ left atrial pressure → pulmonary venous hypertension
- haemoptysis
  - due to pulmonary pressures and vascular congestion
  - may range from pink frothy sputum to sudden haemorrhage secondary to rupture of thin-walled and dilated bronchial veins
- mid-late diastolic murmur (best heard in expiration)
- loud S1
- opening snap
  - indicates mitral valve leaflets are still mobile
- low volume pulse
- malar flush
- atrial fibrillation
  - secondary to ↑ left atrial pressure → left atrial enlargement

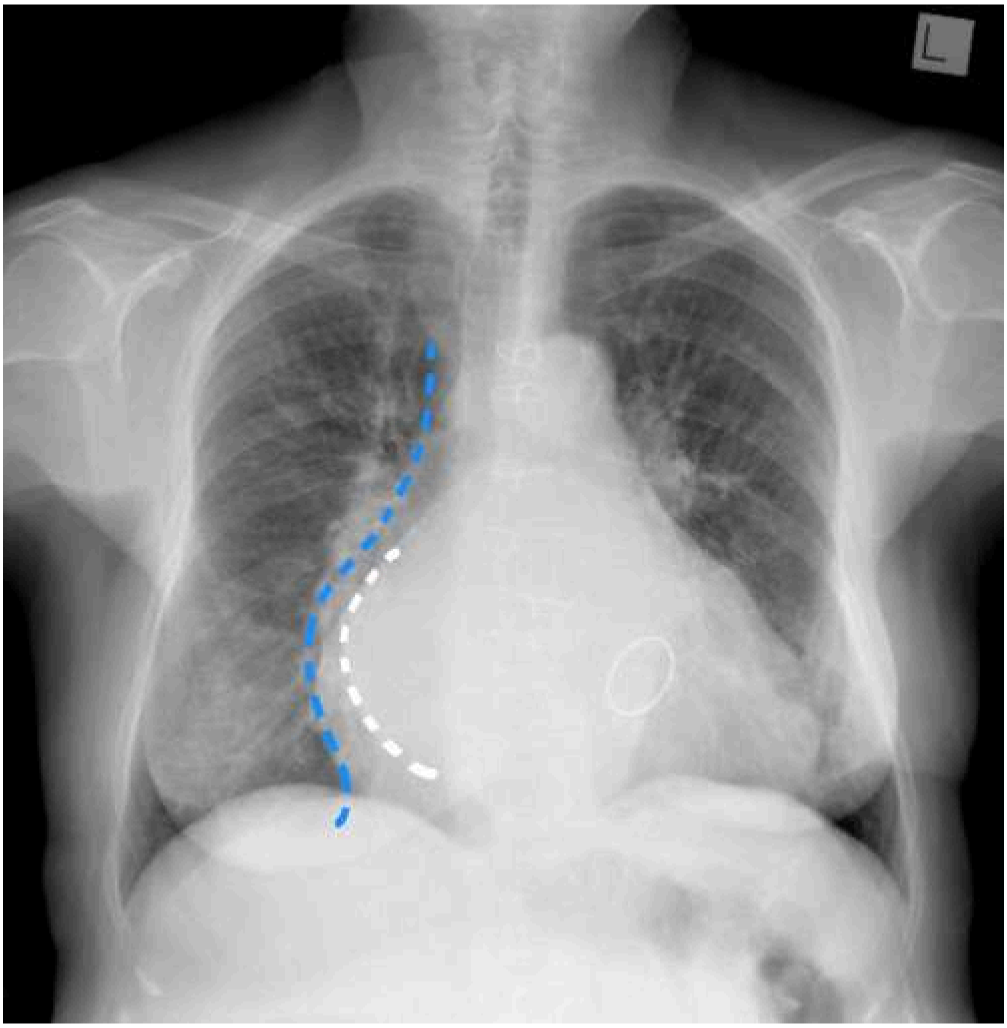
Features of severe MS
- length of murmur increases
- opening snap becomes closer to S2

Chest x-ray
- left atrial enlargement may be seen

Echocardiography
- the normal cross-sectional area of the mitral valve is 4-6 sq cm. A 'tight' mitral stenosis implies a cross-sectional area of < 1 sq cm

Management
- patients with associated atrial fibrillation require anticoagulation
  - currently warfarin is still recommended for patients with moderate/severe MS
  - there is an emerging consensus that direct-acting anticoagulants (DOACs) may be suitable for patients with mild MS who develop atrial fibrillation
- asymptomatic patients
  - monitored with regular echocardiograms
  - percutaneous/surgical management is generally not recommended

- symptomatic patients
    - percutaneous mitral balloon valvotomy
    - mitral valve surgery (commissurotomy, or valve replacement)



© Image used on license from Radiopaedia

Chest x-ray from a patient with mitral stenosis. This patient has had a sternotomy and a prosthetic mitral valve. There is splaying of the carina with elevation of the left main bronchus, a double right heart border and cardiomegaly. The features are those of left atrial enlargement. Although the entire heart is enlarged, a double contour is seen through the right side of the heart. The more medial line is the enlarged left atrium (white dotted line) and the heart border is more lateral (blue dotted line).

+ Q 123

Next question

Textbooks

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Links

Radiopaedia 👍 20 👎 11

Mitral valve stenosis

Suggest link Report broken link

Media

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 4 👎 1

Mitral stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 3 👎 2

Report broken media

Score: **20.3%**

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Question 75 of 129

A 48-year-old female presents with one day history of palpitation and shortness of breath. Her blood pressure is 150/90 mmHg, pulse is 190/min, respiratory rate is 21/min and oxygen saturation is 99% on air. ECG shows irregularly irregular rhythm and intermittent delta waves. Which of the following drug should be used to control the heart rate?

○ Flecainide

○ Digoxin

○ Diltiazem

○ Verapamil

○ Adenosine

Submit answer

Reference ranges

Score: **20.3%**

1 ✗
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Question 75 of 129 ✓

A 48-year-old female presents with one day history of palpitation and shortness of breath. Her blood pressure is 150/90 mmHg, pulse is 190/min, respiratory rate is 21/min and oxygen saturation is 99% on air. ECG shows irregularly irregular rhythm and intermittent delta waves. Which of the following drug should be used to control the heart rate?

Flecainide	77%
Digoxin	4%
Diltiazem	4%
Verapamil	9%
Adenosine	5%

Delta waves suggest this patient has underlying Wolff-Parkinson-White syndrome in addition to atrial fibrillation. According to the NICE guidelines, in this condition, flecainide can be used as an alternative to electrical cardioversion for attempting pharmacological cardioversion. Atrialventricular node-blocking agents such as digoxin, diltiazem or verapamil should not be used. Adenosine is also an atrialventricular node-blocking agent.

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Wolff-Parkinson White ★

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.

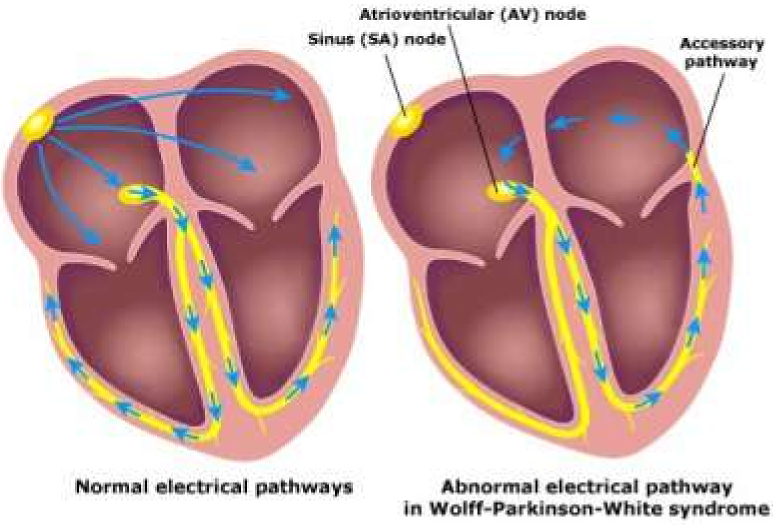
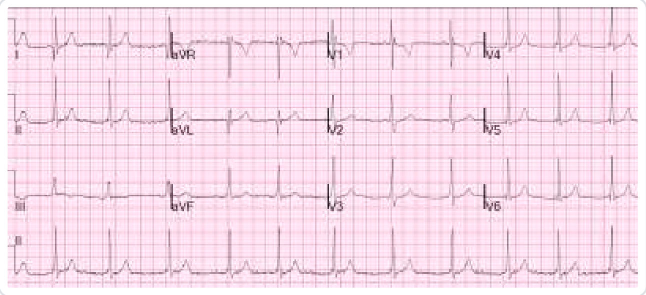

Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway
Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
  - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway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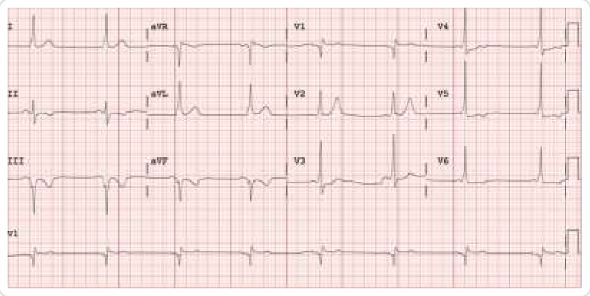
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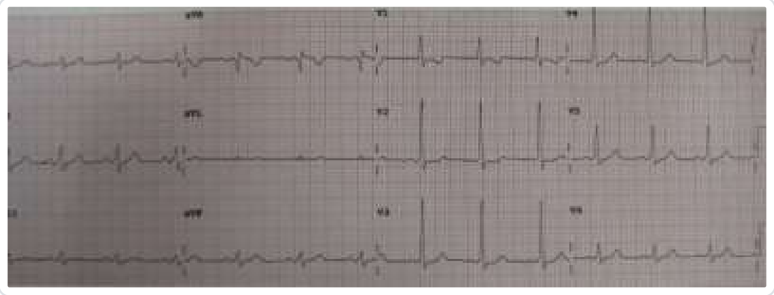
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Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
  - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

AI + Q 123

Next question >

Textbooks

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Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 👍 12 👎 2

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Score: **21.3%**

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Question 76 of 129

A 38-year-old man is referred to the pharmacology clinic for review. He is currently managed for hypertension with three anti-hypertensive agents, (ramipril, amlodipine and indapamide). His GP is concerned as he has still not achieved blood pressure target. On examination his blood pressure is 155/95 mmHg, pulse is 78 beats per minute and regular. There are no heart murmurs or bruits. Abdomen is soft and non tender with no masses and his body mass index is 24 kg/m^2.

Investigations:

Na$^+$	142 mmol/l
K$^+$	3.1 mmol/l
HCO3$^-$	30 mmol/l
Urea	7.0 mmol/l
Creatinine	90 µmol/l

Which of the following is the most likely diagnosis?

- ○ Bartter's syndrome
- ○ Conn's syndrome
- ○ Cushing's syndrome
- ○ Licorice overdose
- ○ Renal artery stenosis

Submit answer

Reference ranges

Score: **21.3%**

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$HCO3^-$	30 mmol/l
Urea	7.0 mmol/l
Creatinine	90 µmol/l

Which of the following is the most likely diagnosis?

Bartter's syndrome	14%
Conn's syndrome	68%
Cushing's syndrome	5%
Licorice overdose	5%
Renal artery stenosis	8%

This patient has resistant hypertension in the presence of hypokalaemia and high bicarbonate, despite initiation of three different anti-hypertensive medications. Although the lack of symptoms or signs of cardiovascular disease doesn't rule out renal artery stenosis completely, it makes it less likely versus Conn's or licorice overdose as a cause of hypertension with hypokalaemia. Licorice overdose is made less likely by the fact that most licorice sold in shops in the UK now has glycyrrhizic acid removed, and this is the active agent which leads to hypokalaemia and hypertension. Conn's is the single most common cause of secondary hypertension, making up between 5 and 10% of cases of hypertension overall.

Bartter's syndrome leads to hypokalaemia with hypotension and is therefore not an appropriate answer here.

👍 👎 💬 Discuss (6) Improve

Next question >

Hypertension: secondary causes ★

It is thought that between 5-10% of patients diagnosed with hypertension have **primary hyperaldosteronism**, including Conn's syndrome. This makes it the single most common cause of secondary hypertension.

Renal disease accounts for a large percentage of the other cases of secondary hypertension. Conditions which may increase the blood pressure include:

- glomerulonephritis
- pyelonephritis
- adult polycystic kidney disease
- renal artery stenosis

Endocrine disorders (other than primary hyperaldosteronism) may also result in increased blood pressure:

- phaeochromocytoma
- Cushing's syndrome
- Liddle's syndrome
- congenital adrenal hyperplasia (11-beta hydroxylase deficiency)
- acromegaly

Drug causes:

- steroids
- monoamine oxidase inhibitors
- the combined oral contraceptive pill

- NSAIDs
- leflunomide

Other causes include:

- pregnancy
- coarctation of the aorta

123

Next question >

Textbooks

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Media

Hypertension

Osmosis - YouTube 3 0

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Score: **21.1%**

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A 28-year-old lady pregnant lady of 37 weeks gestation presented to the Emergency Department a few hours earlier with new onset chest pain. The pain was sharp and made worse with inspiration. She also complained of rapid onset progressively increasing shortness of breath, affecting her ability to complete sentences. She denied the presence of a cough or sputum production and did not suffer from haemoptysis or calf pain. Other than a successful external cephalic version for a breech presentation five days ago and a placenta praevia which spontaneously resolved, her pregnancy was unremarkable. She was in good health with an unremarkable past medical history and normal routine investigations throughout her pregnancy. She smoked 15 cigarettes per day and did not consume alcohol. Her mother suffered from an unexplained deep vein thrombosis when she was 42 years old but otherwise her family history was unremarkable.

Initial examination revealed a heart rate of 122bpm, respiratory rate 24/min, oxygen saturations of 98% on air, a temperature of 37.6ºC and a blood pressure of 112/72 mmHg. She was struggling to complete full sentences. Examination of her cardiorespiratory system revealed good air entry in both bases, a JVP of 3cm, the absence of pedal oedema and soft and non-tender calves. Examination of her gastrointestinal system was unremarkable and ultrasound auscultation revealed the presence of a fetal heartbeat.

As she was being tended to for initial investigations, her condition rapidly deteriorated. Her oxygen saturation dropped to 88% on air, and her blood pressure was recorded as 88/66mmHg. She appeared cool and clammy, and her respiratory rate increased to 32/min. The doctor tending to venepuncture noted the presence of oozing of blood from the wound. She was promptly transferred the intensive care unit and the following investigations were conducted:

Hb	101 g/l
Platelets	75 * 10^9/l
WBC	12.2 * 10^9/l

Na^+	136 mmol/l
K^+	4.8 mmol/l
Urea	14.1 mmol/l
Creatinine	158µmol/l

INR	3.9
APTT	84 s
D-Dimer	2920 ng/ml

Urinalysis: ketones ++, leucocytes/nit/prot/blood/glucose negative

Portable chest x-ray: normal appearance of heart and lung fields

ECG: sinus tachycardia 148bpm, T wave inversion leads V3-V6

Arterial blood gases on air:

pH	7.48
Pa02	5.9 kPa
PaC02	2.2 kPa
BE	1
HC03	24 mmol/l

She was commenced on 15 litres/min oxygen via non-rebreathe mask, and an arterial line and central venous line were sited, as well as the presence of two large bore peripheral cannulae. She was immediately commenced on 2 litres of Hartmann's solution stat.

What is the most likely diagnosis?

○ Pulmonary embolus ×

○ Septic shock ×

○ Peripartum cardiomyopathy ×

○ Aortic dissection ×

○ Amniotic fluid embolus ×

Submit answer

Reference ranges ﹀

Score: **21.1%**

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77	-

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A 28-year-old lady pregnant lady of 37 weeks gestation presented to the Emergency Department a few hours earlier with new onset chest pain. The pain was sharp and made worse with inspiration. She also complained of rapid onset progressively increasing shortness of breath, affecting her ability to complete sentences. She denied the presence of a cough or sputum production and did not suffer from haemoptysis or calf pain. Other than a successful external cephalic version for a breech presentation five days ago and a placenta praevia which spontaneously resolved, her pregnancy was unremarkable. She was in good health with an unremarkable past medical history and normal routine investigations throughout her pregnancy. She smoked 15 cigarettes per day and did not consume alcohol. Her mother suffered from an unexplained deep vein thrombosis when she was 42 years old but otherwise her family history was unremarkable.

Initial examination revealed a heart rate of 122bpm, respiratory rate 24/min, oxygen saturations of 98% on air, a temperature of 37.6ºC and a blood pressure of 112/72 mmHg. She was struggling to complete full sentences. Examination of her cardiorespiratory system revealed good air entry in both bases, a JVP of 3cm, the absence of pedal oedema and soft and non-tender calves. Examination of her gastrointestinal system was unremarkable and ultrasound auscultation revealed the presence of a fetal heartbeat.

As she was being tended to for initial investigations, her condition rapidly deteriorated. Her oxygen saturation dropped to 88% on air, and her blood pressure was recorded as 88/66mmHg. She appeared cool and clammy, and her respiratory rate increased to 32/min. The doctor tending to venepuncture noted the presence of oozing of blood from the wound. She was promptly transferred the intensive care unit and the following investigations were conducted:

Hb	101 g/l
Platelets	75 * 10^9/l
WBC	12.2 * 10^9/l

Na^+	136 mmol/l
K^+	4.8 mmol/l
Urea	14.1 mmol/l
Creatinine	158µmol/l

INR	3.9
APTT	84 s
D-Dimer	2920 ng/ml

Urinalysis: ketones ++, leucocytes/nit/prot/blood/glucose negative

Portable chest x-ray: normal appearance of heart and lung fields

ECG: sinus tachycardia 148bpm, T wave inversion leads V3-V6

Arterial blood gases on air:

pH	7.48
Pa02	5.9 kPa
PaC02	2.2 kPa
BE	1
HC03	24 mmol/l

She was commenced on 15 litres/min oxygen via non-rebreathe mask, and an arterial line and central venous line were sited, as well as the presence of two large bore peripheral cannulae. She was immediately commenced on 2 litres of Hartmann's solution stat.

What is the most likely diagnosis?

Pulmonary embolus	31%
Septic shock	5%
Peripartum cardiomyopathy	6%
Aortic dissection	2%

Amniotic fluid embolus 56%

In this instance, pulmonary embolism would not account for the presence of DIC and there is no evidence of deep vein thrombosis clinically. Septic shock can follow a similar path to amniotic fluid embolus, but in this instance, there is little evidence for a focus of sepsis.

👍 👎 Discuss (8) Improve

Next question >

Amniotic fluid embolism ★

Definition:

- This is when fetal cells/ amniotic fluid enters the mothers bloodstream and stimulates a reaction which results in the signs and symptoms described below.

Epidemiology:

- Rare complication of pregnancy associated with a high mortality rate.Incidence 2/ 100,000 in the U.K .

Aetiology:

- Many risk factors have been associated with amniotic fluid embolism but a clear cause has not been proven. A consistent link has been demonstrated with maternal age and induction of labour. It is widely accepted that maternal circulation must be exposed to fetal cells/ amniotic fluid in order for an amniotic fluid embolism to occur. However the precise underlying pathology of this process which leads to the embolism is not well understood, though suggestions have been made about an immune mediated process.

Clinical presentation:

- The majority of cases occur in labour , though they can also occur during caesarean section and after delivery in the immediate postpartum.
- Symptoms include: chills, shivering, sweating, anxiety and coughing.

- Signs include: cyanosis, hypotension, bronchospasms, tachycardia. arrhythmia and myocardial infarction.

Diagnosis:
- Clinical diagnosis of exclusion, as there are not definitive diagnostic tests.

Management:
- Critical care unit by a multidisciplinary team, management is predominantly supportive

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **20.8%**

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A 77-year-old man has attended the Emergency Department as he is very concerned about a purple lacy rash that has appeared on his arms and legs over the past few days. It is not painful and does not itch. His past medical history includes hypertension, type 2 diabetes, diverticulitis and atrial fibrillation. He takes amlodipine, ramipril and metformin and was started on warfarin one week ago by his GP. Examination confirms a widespread purple mottled rash across all four limbs and the abdomen. His chest is clear, heart sounds are normal and the abdomen is soft, non-tender.

Blood results on admission show:

Hb	119 g/l
MCV	85 fl
Platelets	142 * 10^9/l
WBC	10.1 * 10^9/l
Neuts	4.5 * 10^9/l
Lymphs	1.9 * 10^9/l
Eosinophils	1.5 * 10^9/l

Na^+	142 mmol/l
K^+	4.6 mmol/l
Urea	14.6 mmol/l
Creatinine	169 µmol/l

What is the most likely diagnosis?

○ Amyloidosis

○ Antiphospholipid syndrome

○ Cholesterol atheroemboli

○ Churg-Strauss syndrome ×

○ Focal segmental glomerulosclerosis ×

Submit answer

Reference ranges ⌵

Score: **20.8%**

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Back to top

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Lymphs	1.9 * 10^9/l
Eosinophils	1.5 * 10^9/l

Na^+	142 mmol/l
K^+	4.6 mmol/l
Urea	14.6 mmol/l
Creatinine	169 µmol/l

What is the most likely diagnosis?

Amyloidosis	4%
Antiphospholipid syndrome	6%
Cholesterol atheroemboli	80%

Churg-Strauss syndrome	8%
Focal segmental glomerulosclerosis	2%

The purple, lacy, non-itchy rash described here is livedo reticularis. Antiphospholipid syndrome, Churg-Strauss syndrome and cholesterol atheroemboli can all cause livedo reticularis

Churg-Strauss and cholesterol atheroemboli could both result in raised eosinophils as seen here. However, this patient was started on warfarin a week ago by his GP which a recognised precipitant of cholesterol atheroemboli which makes it the most likely diagnosis here.

Discuss (6) Improve

Next question

Cholesterol embolisation

Overview

- cholesterol emboli may break off causing renal disease
- the majority of cases are secondary to vascular surgery or angiography. Other causes include severe atherosclerosis, particularly in large arteries such as the aorta

Features

- eosinophilia
- purpura
- renal failure
- livedo reticularis

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **20.5%**

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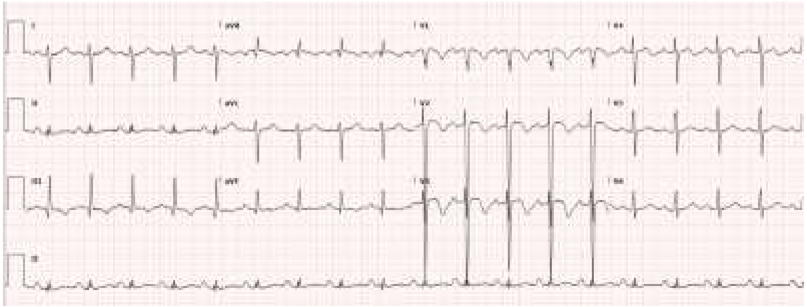
Question 79 of 129

A man in his 50's is brought to the emergency department by ambulance and was found collapsed in the street. His vital signs are as follows: Blood pressure 90/60 mmHg, pulse 120 bpm, respirations 25 bpm and temperature 37.9 ºC.

The 12 lead ECG showed:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely cause of the patient's presentation?

- ○ Non-ST elevation myocardial infarction ×
- ○ Pulmonary embolism ×
- ○ ST elevation myocardial infarction ×
- ○ Severe hypokalaemia ×
- ○ Takotsubo cardiomyopathy ×

Submit answer

Reference ranges

Score: **20.5%**

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Question 79 of 129

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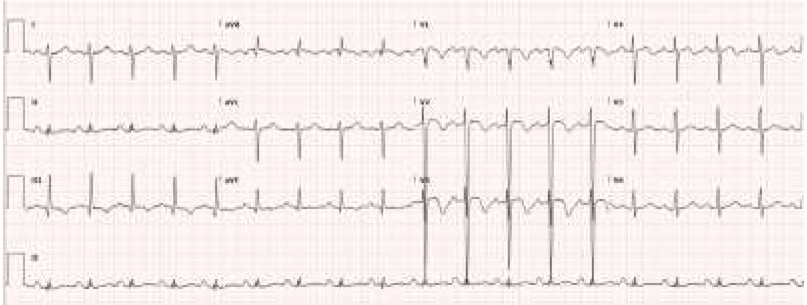

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What is the most likely cause of the patient's presentation?

Non-ST elevation myocardial infarction	13%
Pulmonary embolism	65%
ST elevation myocardial infarction	10%
Severe hypokalaemia	8%
Takotsubo cardiomyopathy	4%

The ECG shows sinus tachycardia with right axis deviation, P pulmonale, Q waves in lead III and inverted T waves in the right precordial leads (V1-V3) and lead III.

Pulmonary embolism is the correct answer. Although the ECG is neither sensitive nor specific for pulmonary embolism, the classical findings of S1Q3T3 highly suggest right ventricular strain. Hence, the clinical picture is highly suspicious for pulmonary embolism, and the patient should promptly have an urgent CT pulmonary angiogram and thrombolysis if indicated. Other acute causes for right ventricular strain, like pneumothorax, upper airway obstruction and exacerbation of asthma/COPD, should also be considered in the differentials.

Non ST elevation myocardial infarction is incorrect. ECG changes like ST depression or T wave inversion in NSTEMI are often non-specific. If new, the T-wave inversions in this patient may suggest acute coronary syndrome, particularly right ventricular infarction, given the right ventricular strain pattern; however, NSTEMI is unlikely to cause collapse. The classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

ST elevation myocardial infarction is incorrect. Classically, STEMI is diagnosed if there is > 1-2 mm ST elevation in 2 contiguous leads or a new left bundle branch block (LBBB). Both of these are not found in this patient's ECG.

Severe hypokalaemia is incorrect. Although hypokalaemia may present with T-wave inversions in the precordial leads, there are no ST depression or U waves commonly seen in hypokalaemia. While hypokalaemia can cause muscle tetany, paralysis and respiratory failure, it is unlikely to cause collapse.

Takotsubo cardiomyopathy is incorrect. ECG changes like T wave inversion in Takotsubo cardiomyopathy are often non-specific. Moreover, the classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

👍 👎 Discuss (2) Improve

Next question ›

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

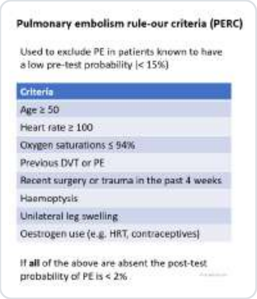
The relative frequency of common clinical signs is shown below:
- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)
- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
  - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

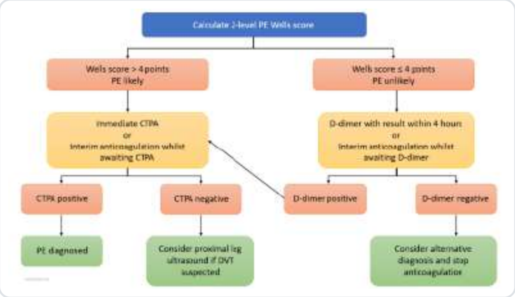
Clinical probability simplified scores
- PE likely - more than 4 points
- PE unlikely - 4 points or less

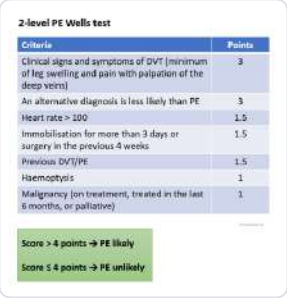
If a PE is 'likely' (more than 4 points)
- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
  - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin

    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
    - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points
PE likely
Wells score ≤ 4 points
PE unlikely
Immediate CTPA
or
Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours
or
Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging

and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

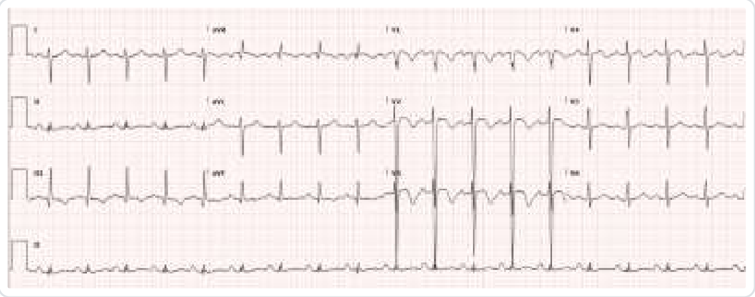
Some other points

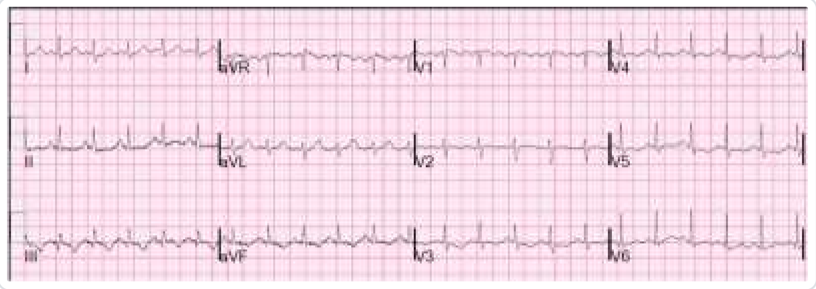
D-dimers

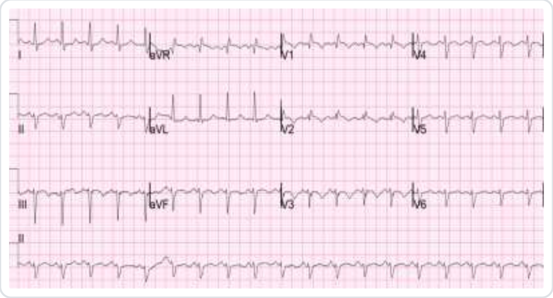
- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen



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II

Chest x-ray

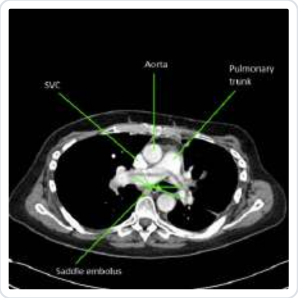
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

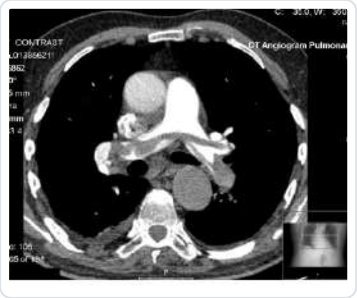
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


SVC
Aorta
Pulmonary trunk
Saddle embolus


CONTRAST
CT Angiogram Pulmonar

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

+ Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

MDcalc 👍 4 👎 0

PERC Rule for Pulmonary Embolism

NICE 👍 2 👎 1

2020 Venous thromboembolism guidelines

Life in the Fast Lane 👍 1 👎 1

The ECG in Pulmonary Embolism

MDcalc 👍 1 👎 1

Wells' Criteria for Pulmonary Embolism

Suggest link Report broken link

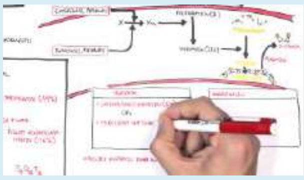
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

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A 50-year-old woman was referred to the clinic with a 6-month history of shortness of breath. She gave a history of progressive exertional breathlessness and now could only walk a couple of hundred meters. In addition she also noted swelling of her ankles that started 3 weeks ago and had experienced 1 episodes of loss of consciousness. She had been a wheezy child but had grown out of it. She was a non-smoker and drank approximately 5 units of alcohol per week. She took no regular medications and had no known allergies. Her General Practitioner had prescribed salbutamol and beclomethasone inhalers for her but this had made no difference.

On examination her heart rate was 86 beats per minute, blood pressure 135/90 mmHg, respiratory rate 20 breaths per minute and oxygen saturations were 93% on room air.

Initial investigations are shown below:

Full blood count, urea and electrolytes, liver function tests: normal
Autoantibody screen: negative

Chest x-ray: clear lung fields, no cardiomegaly

Lung function tests:
-forced expiratory volume in 1-second 2.8L (115% predicted)
-forced vital capacity 3.2L (120% predicted)
-total lung capacity 5.8L (120% predicted)
-carbon monoxide diffusion in the lungs 92%

Transthoracic echocardiogram:
-left ventricle: normal size, ejection fraction 65% (>55)
-normal aortic and pulmonary valves
-right ventricle dilated
-tricuspid regurgitation (mild)
-pulmonary artery systolic pressure: 55 mmHg

CT scan on chest: normal

Ventilation-perfusion (V/Q) scan: low probability for thromboembolic disease

What is the appropriate next investigation?

- ○ Bronchoscopy and bronchoalveolar lavage ×
- ○ Cardiac catheterisation ×
- ○ CT guided lung biopsy ×
- ○ CT pulmonary angiography ×
- ○ Genetic testing for cystic fibrosis ×

Submit answer

Reference ranges

Score: **20.3%**

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-pulmonary artery systolic pressure: 55 mmHg

CT scan on chest: normal

Ventilation-perfusion (V/Q) scan: low probability for thromboembolic disease

What is the appropriate next investigation?

Bronchoscopy and bronchoalveolar lavage	3%
Cardiac catheterisation	76%
CT guided lung biopsy	2%
CT pulmonary angiography	15%
Genetic testing for cystic fibrosis	3%

The history and investigations in this case suggest a diagnosis of idiopathic pulmonary arterial hypertension (IPAH).

This condition is more common in women and commonly presents with progressive exertional breathlessness, lethargy, fatigue or syncope. On examination a loud pulmonary heart sound may be heard and eventually murmurs of pulmonary and tricuspid regurgitation may also be heard. As the disease progresses and right heart failure develops examination may reveal an elevated jugular venous pulse, hepatomaegaly, ascities and peripherial oedema.

Aside from idiopathic common causes include congenital/acquired cardiac lesions. Chronic obstructive pulmonary disease (COPD) of other lung pathology, pulmonary thromboembolic disease and vasculitic disorders.

ECHO is a good screening test but to confirming the diagnosis requires the gold standard investigation, which is cardiac catheterisation. Cardiac catheterisation will show the oxygen saturations in the veins and chambers of the heart and allow for accurate measurement of right atrial, ventricular and pulmonary artery pressures and pulmonary capillary wedge pressure, pulmonary vascular resistance and response to acute vasodilators (important implications for treatment).

The definition of IPAH includes: mean pulmonary artery pressure > 25 mmHg, pulmonary artery wedge pressure <= 15 mmHg and pulmonary vascular resistance > 3 Wood units.

Discuss (4) | Improve

Next question ›

Pulmonary arterial hypertension

Pulmonary arterial hypertension (PAH) may be defined as a resting mean pulmonary artery pressure of >= 20 mmHg. Endothelin is thought to play a key role in the pathogenesis of PAH. It is more common in females and typically presents between the ages of 30-50 years. Pulmonary hypertension may of course develop secondary to chronic lung diseases such as COPD - PAH is diagnosed in the absence of this although certain factors increase the risk, including HIV, cocaine and anorexigens (e.g. fenfluramine). Around 10% of cases are inherited in an autosomal dominant fashion.

Features

- progressive exertional dyspnoea is the classical presentation
- other possible features include exertional syncope, exertional chest pain and peripheral oedema
- cyanosis
- clinical signs:
    - right ventricular heave: indicating right ventricular hypertrophy or dilatation
    - loud P2: early in the disease reflects increased pulmonary artery pressure and may be accompanied by a palpable P2 in severe cases. With advanced PAH there may be right ventricular failure leading to a soft S2
    - raised JVP with prominent 'a' waves: reflects increased resistance to right atrial emptying due to elevated right ventricular end-diastolic pressure
    - tricuspid regurgitation

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that **acute vasodilator testing** is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide.

If there is a positive response to acute vasodilator testing (a minority of patients)

- oral calcium channel blockers

If there is a negative response to acute vasodilator testing (the vast majority of patients)

- prostacyclin analogues: treprostinil, iloprost
- endothelin receptor antagonists
    - non-selective: bosentan
    - selective antagonist of endothelin receptor A: ambrisentan
- phosphodiesterase inhibitors: sildenafil

Patients with progressive symptoms should be considered for a heart-lung transplant.

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 12 13

2015 Guidelines for the diagnosis and treatment of pulmonary hypertension

Medcomic 👍 14 👎 8

Pulmonary hypertension

Respiratory Medicine journal 👍 13 👎 7

Pulmonary vasodilator testing and use of calcium channel blockers in pulmonary arterial hypertension

Suggest link Report broken link

Media

Pulmonary hypertension

Osmosis - YouTube 👍 1 👎 4

Report broken media

Score: **20%**

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Question 81 of 129

A 55-year-old man presents for review in cardiology clinic for his angina pectoris. He developed exertional chest pain two years ago and has been managed medically since that point with GTN, aspirin, atorvastatin and atenolol. He finds that he is unable to manage his angina and frequently needs the use of his GTN spray, which causes him headaches. He has a past medical history of gout and depression as well. He works as a paralegal and has quit smoking successfully five months ago. He is keen to better control the pain.

What is the most appropriate management option?

○ Add on nicorandil ×

○ Add on long-acting nitrite ×

○ Add on ivabradine ×

○ Refer for coronary arterial bypass graft ×

○ Add on calcium channel blocker ×

Submit answer

Reference ranges

Score: **20%**

1 ✗
2 ✓
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What is the most appropriate management option?

Add on nicorandil	7%
Add on long-acting nitrite	8%
Add on ivabradine	3%
Refer for coronary arterial bypass graft	2%
Add on calcium channel blocker	79%

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

Important for me Less important

The correct answer is to add on a calcium channel blocker to his regular medications. He has been managed on monotherapy with a beta-blocker and prophylactic medication to prevent further ischaemic heart disease. Since first line monotherapy has failed, it should be combined with a calcium channel blocker following full up-titration of the monotherapy medication. If a combination of beta-blocker and calcium channel blocker fail to control symptoms then a long-acting nitrate, ivabradine, nicorandil or ranolazine can be added. Once medical management fails, surgery should be considered for further management.

Source:

'Unstable angina and NSTEMI: early management' Clinical guideline [CG94]. The National Institute for Health and Care Excellence, March 2010.

👍 👎 Discuss (4) Improve

Next question >

Angina pectoris: drug management

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil

    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 22 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

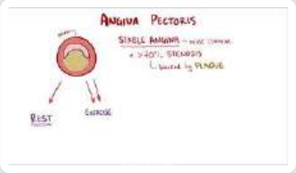
Suggest link Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

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Score: **19.8%**

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Question 82 of 129

A 34-year-old woman comes to haematology clinic. She presented with a shortness of breath three days ago to a local emergency department. She underwent a CT pulmonary angiogram scan which demonstrated a pulmonary embolus. She was discharged with injections of low molecular weight heparin at treatment dose with a view of attending the anticoagulation clinic to start warfarin. She has no past medical history or allergies and only takes Microgynon as an oral contraceptive.

She is not pregnant and agrees to start warfarin.

What should be done with her heparin whilst starting warfarin?

- ○ Switch to prophylactic dose until INR is known to be above 2 ×
- ○ Continue for one week whilst on warfarin ×
- ○ Stop same day as starting warfarin ×
- ○ Continue alongside warfarin for two weeks ×
- ○ Continue until INR is known to be above 2 ×

Submit answer

Reference ranges

Score: **19.8%**

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Question 82 of 129 ✓

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What should be done with her heparin whilst starting warfarin?

Switch to prophylactic dose until INR is known to be above 2	5%
Continue for one week whilst on warfarin	13%
Stop same day as starting warfarin	3%
Continue alongside warfarin for two weeks	6%
Continue until INR is known to be above 2	73%

The correct answer is to continue until INR is known to be above 2. This is a patient is started on warfarin whilst being covered with low-molecular weight heparin for a pulmonary embolism. As she has not contraindications for warfarin, such as pregnancy or malignancy, warfarin should be started with a target INR of 2.5 (acceptable range 2-3). Starting warfarin can be variable and there are several protocols to achieve hospital induction, but whilst the patient has not yet been known to be at therapeutic anticoagulation, low-molecular weight heparin should be continued until a therapeutic INR is confirmed. Once this is achieved, the low-molecular weight heparin can be stopped.

Discuss (4) | Improve

Next question ›

Warfarin ★

Warfarin is an oral anticoagulant that was used first-line for many years in both the management of venous thromboembolism and reducing stroke risk in patients with atrial fibrillation. It has now been largely superseded by the use of direct oral anticoagulants (DOACs) which do not require the same level of monitoring as warfarin.

Mechanism of action

- inhibits epoxide reductase preventing the reduction of vitamin K to its active hydroquinone form
- this in turn acts as a cofactor in the carboxylation of clotting factor II, VII, IX and X (mnemonic = 1972) and protein C.

Indications

- mechanical heart valves
  - target INR depends on the valve type and location
  - mitral valves generally require a higher INR than aortic valves.
- second-line after DOACs:
  - venous thromboembolism: target INR = 2.5, if recurrent 3.5
  - atrial fibrillation, target INR = 2.5

Monitoring

- patients are monitored using the INR (international normalised ratio), the ratio of the prothrombin time for the patient over the normal prothrombin time.
- warfarin has a long half-life and achieving a stable INR may take several days
- there are a variety of loading regimes and computer software is now often used to alter the dose

Factors that may potentiate warfarin

- liver disease
- P450 enzyme inhibitors, e.g.: amiodarone, ciprofloxacin
- cranberry juice
- drugs which displace warfarin from plasma albumin, e.g. NSAIDs
- inhibit platelet function: NSAIDs

Side-effects

- haemorrhage
- teratogenic, although can be used in breastfeeding mothers
- skin necrosis
  - when warfarin is first started biosynthesis of protein C is reduced
  - this results in a temporary procoagulant state after initially starting warfarin, normally avoided by concurrent heparin administration
  - thrombosis may occur in venules leading to skin necrosis
- purple toes

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

British Journal of Hematology 👍 5 👎 9

Peri-Operative Management of Anticoagulation and Antiplatelet Therapy 2016

BNF 👍 7 👎 7

Oral anticoagulants

DermnetNZ 👍 16 👎 7

Warfarin induced skin necrosis

BNF 👍 10 👎 4

Warfarin

British Committee for Standards in Haematology 👍 24 👎 1

Guidelines on oral anticoagulation (warfarin): fourth edition - 2011 update

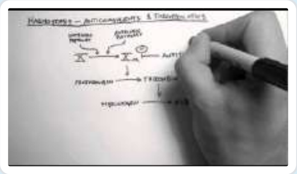
Suggest link

Report broken link

Media

Anticoagulants and antiplatelets

Speed Pharmacology - YouTube 👍 4 👎 0



Haemostasis 3 - Anticoagulants & Thrombolytics

Handwritten Tutorials - YouTube 👍 2 👎 1

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Score: **20.7%**

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An 84-year-old female presents with a three-day history of constant chest discomfort associated with productive green sputum. On admission to the emergency department, a temperature of 38.2 degrees is recorded and she reports a recent history of 'shivering and shaking' over the past 24 hours. She reports no nausea or vomiting and was last well 72 hours ago, when she was living alone with BD carers. She has a known history of bronchiectasis, angina, hypertension, type 2 diabetes mellitus and hypertrophic obstructive cardiomyopathy.

On examination, you note bibasal coarse inspiratory crackles and an ejection systolic murmur. She has noted no deterioration in her exercise tolerance of 150 yards on flat ground. Her blood pressure is 140/85 mmHg heart rate 90/min and regular, Sats 94% on air, respiratory rate 26/min. Her ECG demonstrates ST depression and T wave inversion in V4 to V6, S wave in V1 and R wave in V5 add to 55 mm. You have no previous ECGs to compare this to. Her blood tests are as follows:

Hb	137 g/l
Platelets	402 * 10^9/l
WBC	18.3 * 10^9/l
Neutrophils	16.3 * 10^9/l

Na^+	144 mmol/l
K^+	4.3 mmol/l
Urea	6.2 mmol/l
Creatinine	90 µmol/l
CRP	145 mg/l
Troponin < 0.03 (normal < 0.03)	

Her chest radiograph demonstrates left basal shadowing on a background of bibasal tramlining and fibrotic changes. Previous sputum cultures demonstrate pseudomonas colonisation. New sputum and blood cultures have been taken. What is the most appropriate treatment?

○ Intravenous tazocin

○ Intravenous tazocin and acute coronary syndrome treatment

○ Acute coronary syndrome treatment only

○ Oral amoxicillin only

○ Oral amoxicillin and acute coronary syndrome treatment

Submit answer

Reference ranges

Score: **20.7%**

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Her chest radiograph demonstrates left basal shadowing on a background of bibasal tramlining and fibrotic changes. Previous sputum cultures demonstrate pseudomonas colonisation. New sputum and blood cultures have been taken. What is the most appropriate treatment?

Intravenous tazocin	64%
Intravenous tazocin and acute coronary syndrome treatment	22%
Acute coronary syndrome treatment only	1%
Oral amoxicillin only	9%
Oral amoxicillin and acute coronary syndrome treatment	3%

The patient is elderly and has a number of comorbidities. The presenting condition is clearly infective exacerbation of bronchiectasis on a background of previous pseudomonal colonisation. As a result, oral amoxicillin is insufficient. The second question is regarding these abnormal ECG changes on a background of no cardiac chest pain and a negative first troponin. The key is to notice the history of HOCM with an ejection systolic murmur, suggesting a degree of left ventricular outlet obstruction. Her ECG demonstrates LV hypertrophy and the ST segment and T wave inversion are suggestive of LV strain, an expected pattern for patients with HOCM. No ACS treatment is thus necessary.

Discuss (5) Improve

Next question >

Hypertrophic obstructive cardiomyopathy: features ★

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. The estimated prevalence is 1 in 500. HOCM is important as it is the most common cause of sudden cardiac death in the young.

Pathophysiology

- the most common defects involve a mutation in the gene encoding β-myosin heavy chain protein or myosin-binding protein C
- results in predominantly diastolic dysfunction

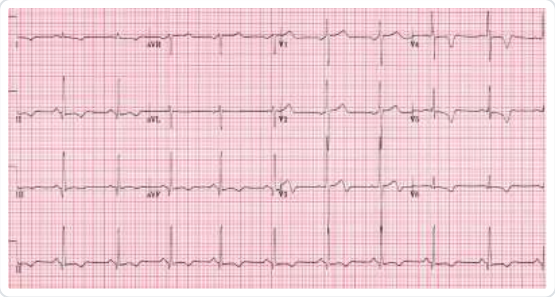
    - left ventricle hypertrophy → decreased compliance → decreased cardiac output
- characterized by myofibrillar hypertrophy with chaotic and disorganized fashion myocytes ('disarray') and fibrosis on biopsy

Features
- often asymptomatic
- exertional dyspnoea
- angina
- syncope
    - typically following exercise
    - due to subaortic hypertrophy of the ventricular septum, resulting in functional aortic stenosis
- sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure
- jerky pulse, large 'a' waves, double apex beat
- systolic murmurs
    - ejection systolic murmur: due to left ventricular outflow tract obstruction. Increases with Valsalva manoeuvre and decreases on squatting
    - pansystolic murmur: due to systolic anterior motion of the mitral valve → mitral regurgitation

Associations
- Friedreich's ataxia
- Wolff-Parkinson White

Echo findings - mnemonic - MR SAM ASH
- mitral regurgitation (MR)
- systolic anterior motion (SAM) of the anterior mitral valve leaflet
- asymmetric hypertrophy (ASH)

ECG
- left ventricular hypertrophy
- non-specific ST segment and T-wave abnormalities, progressive T wave inversion may be seen
- deep Q waves
- atrial fibrillation may occasionally be seen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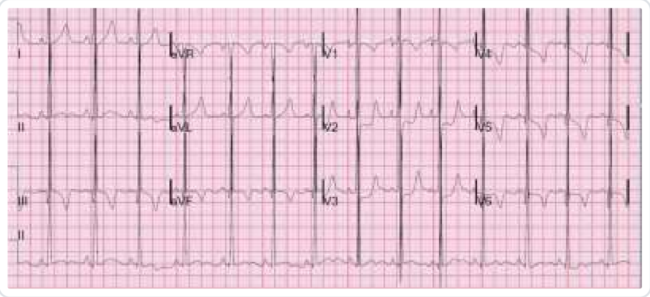
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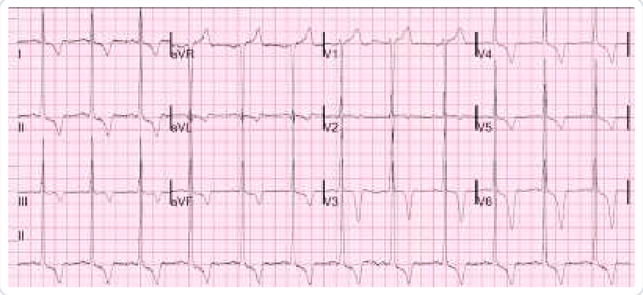
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123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology (ESC)

👍 9 👎 8

2014 ESC HCOM Guidelines

RCP 👍 16 👎 4

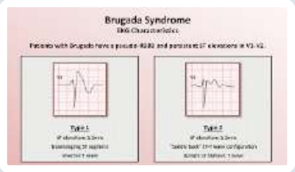
Investigation and treatment of hypertrophic cardiomyopathy

Suggest link Report broken link

Media

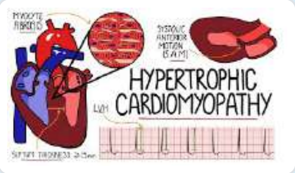
Hypertrophic cardiomyopathy

Osmosis - YouTube 👍 0 👎 0


Brugada Syndrome

Advanced EKGs - Sudden Cardiac Death (Hypertrophic cardiomyopathy, ARVD, Brugada syndrome, and CPVT)

Strong Medicine - YouTube 👍 6 👎 1


HYPERTROPHIC
CARDIOMYOPATHY
SYSTOLIC ANTERIOR MOTION (SAM)
LVH

Hypertrophic Cardiomyopathy Made Easy

Rhesus Medicine - YouTube 👍 1 👎 1

Hypertrophic Cardiomyopathy heart sounds - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 1 👎 1

CARDIOLOGY
HYPERTROPHIC
CARDIOMYOPATHY

Hypertrophic obstructive cardiomyopathy

Osmosis - YouTube 👍 0 👎 4

Report broken media

Score: **20.5%**

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Question 84 of 129

A 54-year-old male business executive is referred to you after a heart murmur is detected at a medical examination he received after he transferred to a new company. He has no known past medical history or family history. He is well and leads an active lifestyle. On examination, you note a pansystolic murmur in the apex. Chest auscultation is unremarkable. An ECG demonstrates sinus rhythm at 64 beats/minute with no changes suggestive of ventricular hypertrophy. A transthoracic echocardiogram demonstrated good views with severe mitral regurgitation, preserved left ventricular function (EF 85%) and pulmonary arterial systolic pressure of 15 mmHg. Which of the following is appropriate management?

- ○ Mitral valve replacement ×
- ○ Mitral valve repair ×
- ○ Percutaneous mitral valve repair (Mitraclip) ×
- ○ Infective endocarditis prophylaxis and 6 monthly echocardiogram ×
- ○ 6 monthly echocardiogram ×

Submit answer

Reference ranges

Score: **20.5%**

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Question 84 of 129 ✓

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Mitral valve replacement	21%
Mitral valve repair	8%
Percutaneous mitral valve repair (Mitraclip)	10%
Infective endocarditis prophylaxis and 6 monthly echocardiogram	5%
6 monthly echocardiogram	55%

The patient has severe chronic non-ischaemic mitral regurgitation that has not decompensated. The aetiology is unclear: primary causes include mitral valve prolapse, flail leaflet or rheumatic heart disease. Secondary causes include cardiomyopathy or coronary artery disease. The patient does not meet indications for intervention on the mitral valve, which include symptoms, left ventricular dysfunction, pulmonary hypertension, new atrial fibrillation and dilated left ventricle. It is uncommon for intervention to take place without any of the indications mentioned above. Instead, serial monitoring with 6 monthly echocardiograms is appropriate. Infective endocarditis prophylaxis is no longer indicated for patients in the absence of a prosthetic valve repair or replacement.

Percutaneous mitral valve repair is an emerging technique that is available to patients considered too high risk for mitral valve surgery, which uses a device to individually approximate the regurgitant leaflet. Comparing Mitraclip with mitral valve replacement or repair in severe mitral regurgitation patients with NYHA III/IV heart failure symptoms, one-year and four-year survival were similar with similar improvements in symptoms, LV size and function. However, Mitraclip patients demonstrated significantly higher incidence of requiring further surgery but significantly lower risks of major adverse events post-procedure.

1. Feldman T, Foster E, Glower DD et al. Percutaneous repair or surgery for mitral regurgitation. N Engl J Med. 2011;364(15):1395

👍 👎 Discuss (10) Improve

Next question >

Mitral regurgitation

Also known as mitral insufficiency, mitral regurgitation (MR) occurs when blood leaks back through the mitral valve on systole. It is the second most common valve disease after aortic stenosis. The mitral valve is located between the left atrium and ventricle, and regurgitation leads to a less efficient heart as less blood is pumped through the body with each contraction. This said, MR is common in otherwise healthy patients to a trivial degree and does not need treatment.

As the degree of regurgitation becomes more severe, the body's oxygen demands may exceed what the heart can supply and as a result, the myocardium can thicken over time. While this may be benign initially, patients may find themselves increasingly fatigued as a thicker myocardium becomes less efficient, and eventually go into irreversible heart failure.

Risk factors

- Female sex
- Lower body mass
- Age
- Renal dysfunction
- Prior myocardial infarction
- Prior mitral stenosis or valve prolapse

- Collagen disorders e.g. Marfan's Syndrome and Ehlers-Danlos syndrome

Causes

- Following coronary artery disease or post-MI: if the papillary muscles or chordae tendinae are affected by a cardiac insult, mitral valve disease may ensue as a result of damage to its supporting structures.
- Mitral valve prolapse: Occurs when the leaflets of the mitral valve is deformed so the valve does not close properly and allows for backflow. Most patients with this have a trivial degree of mitral regurgitation.
- Infective endocarditis: When vegetations from the organisms colonising the heart grow on the mitral valve, it is prevented from closing properly. Patients with abnormal valves are more likely to develop endocarditis as opposed to their peers.
- Rheumatic fever: While this is uncommon in developed countries, rheumatic fever can cause inflammation of the valves and therefore result in mitral regurgitation.
- Congenital

Symptoms

- Most patients with MR are asymptomatic, and patients suffering from mild to moderate MR may stay largely asymptomatic indefinitely. Symptoms tend to be due to failure of the left ventricle, arrhythmias or pulmonary hypertension. This may present as fatigue, shortness of breath and oedema.

Signs

- The murmur heard on auscultation of the chest is typically a pansystolic murmur described as 'blowing'. It is heard best at the apex and radiating into the axilla. S1 may be quiet as a result of incomplete closure of the valve. Severe MR may cause a widely split S2

Investigations

- ECG may show a broad P wave, indicative of atrial enlargement
- Cardiomegaly may be seen on chest x-ray, with an enlarged left atrium and ventricle
- Echocardiography is crucial to diagnosis and to assess severity

Treatment options

- Medical management in acute cases involves nitrates, diuretics, positive inotropes and an intra-aortic balloon pump to increase cardiac output
- If patients are in heart failure, ACE inhibitors may be considered along with beta-blockers and spironolactone
- In acute, severe regurgitation, surgery is indicated
- The evidence for repair over replacement is strong in degenerative regurgitation, and is demonstrated through lower mortality and higher survival rates
- When this is not possible, valve replacement with either an artificial valve or a pig valve is considered

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

MITRAL
REGURGITATION
(INSUFFICIENCY)

Mitral regurgitation

Armando Hasudungan - YouTube 👍 5 👎 0

Mitral valve disease (regurgitation, stenosis)

Osmosis - YouTube 👍 1 👎 0

Mitral regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 1

Report broken media

Score: **21.4%**

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Question 85 of 129

A 75-year-old male presents with increasing chest pain on exertion. He was first diagnosed with stable angina 7 years ago. His past medical history includes hypercholesterolaemia and hypertension. His drugs history includes bisoprolol and nifedipine, which he has been taking for the past 3 years. A recent angiogram was arranged as an outpatient by his GP, demonstrating stenoses in his left circumflex artery, distal right coronary artery and mid-left anterior descending artery. What is the most appropriate long-term treatment of choice for his angina?

- ○ Coronary artery bypass graft ×
- ○ Percutaneous coronary intervention with 3 stents ×
- ○ Addition of ivabradine ×
- ○ Addition of nicorandil ×
- ○ Addition of ranolazine ×

Submit answer

Reference ranges

Score: **21.4%**

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Question 85 of 129 ✓

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Coronary artery bypass graft	72%
Percutaneous coronary intervention with 3 stents	19%
Addition of ivabradine	2%
Addition of nicorandil	4%
Addition of ranolazine	3%

NICE guidelines recommend the use of no more than 2 antianginals prior to consideration of reperfusion therapies. In the case of this gentleman, the consideration would be whether CABG or PCI would offer greater benefits: although the risks of recurrent MIs and stroke are similar for both PCI and CABG at one year, patients with complex anatomy, triple vessel disease or proximal left mainstem disease report better long-term survival and freedom from MI is greater with CABG. CABG is thus favoured to PCI in this patient according to American Heart Association and European Society of Cardiology guidelines.

Discuss (4) Improve

Next question

Angina pectoris: drug management

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
  - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
  - a long-acting nitrate
  - ivabradine
  - nicorandil
  - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 22 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

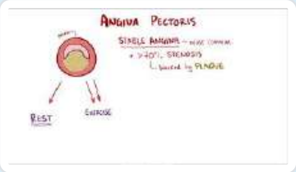
Suggest link Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
STENOSIS
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **22.4%**

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Back to top

A 70-year-old male presents back to hospital 3 weeks after undergoing a primary percutenaous coronary intervention for anterior STEMI. He reports 24 hour history of increasing shortness of breath, fever over 39 degrees and pleuritic chest pain, different from the pain he reported when he initially presented with his ischaemic chest pain. On examination, his calves are soft, his heart sounds are normal with no added sounds and his lungs are clear on auscultation. His serum tests reveals a mildly raised troponin and ECG that demonstrates ST elevation in V2 to V4 but no reciprocal change. Angiography was performed again, demonstrating good radiological flow with no evidence of stent thrombosis. An transthoracic echocardiogram demonstrates a mild to moderate pericardial effusion with no tamponade. What is the optimal initial treatment?

- ○ Aspirin, clopidogrel, low molecular weight heparin as per acute coronary syndrome protocol
- ○ Fondaparinux
- ○ Bivaluridin
- ○ Oral prednisolone
- ○ Oral ibuprofen

Submit answer

Reference ranges

Score: **22.4%**

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Question 86 of 129 ✗

A 70-year-old male presents back to hospital 3 weeks after undergoing a primary percutenaous coronary intervention for anterior STEMI. He reports 24 hour history of increasing shortness of breath, fever over 39 degrees and pleuritic chest pain, different from the pain he reported when he initially presented with his ischaemic chest pain. On examination, his calves are soft, his heart sounds are normal with no added sounds and his lungs are clear on auscultation. His serum tests reveals a mildly raised troponin and ECG that demonstrates ST elevation in V2 to V4 but no reciprocal change. Angiography was performed again, demonstrating good radiological flow with no evidence of stent thrombosis. An transthoracic echocardiogram demonstrates a mild to moderate pericardial effusion with no tamponade. What is the optimal initial treatment?

Aspirin, clopidogrel, low molecular weight heparin as per acute coronary syndrome protocol	5%
Fondaparinux	2%
Bivaluridin	1%
Oral prednisolone	18%
Oral ibuprofen	74%

The diagnosis is Dresslers syndrome, an inflammatory post-cardiac injury complication, characterised by high fever, chest pain with a possible pleural or pericardial rub and raised inflammatory markers. It is distinguished by late presentation compared to acute reperfusion injury, which normally occurs days to weeks after the acute MI. Dresslers syndrome is thus thought to be immune mediated instead. Most patients symptoms resolve completely with oral NSAIDs but a few refractory cases require corticosteroid therapy.

Discuss (18) | Improve

Next question ›

Myocardial infarction: complications

Patients are at risk of a number of immediate, early and late complications following a myocardial infarction (MI).

Cardiac arrest

This most commonly occurs due to patients developing ventricular fibrillation and is the most common cause of death following a MI. Patients are managed as per the ALS protocol with defibrillation.

Cardiogenic shock

If a large part of the ventricular myocardium is damaged in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock. This is difficult to treat. Other causes of cardiogenic shock include the 'mechanical' complications such as left ventricular free wall rupture as listed below. Patients may require inotropic support and/or an intra-aortic balloon pump.

Chronic heart failure

As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop diuretics such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias

Ventricular fibrillation, as mentioned above, is the most common cause of death following a MI. Other common arrhythmias including ventricular tachycardia.

Bradyarrhythmias

Atrioventricular block is more common following inferior myocardial infarctions.

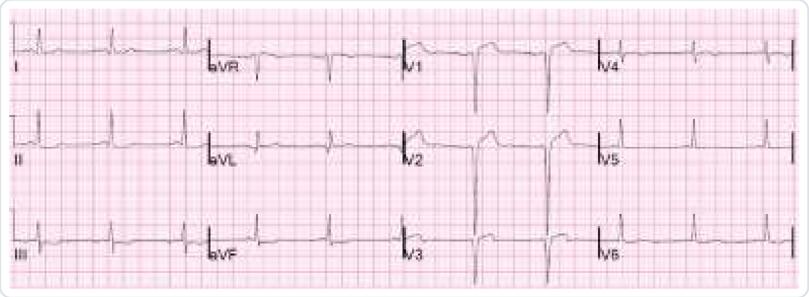
Pericarditis

Pericarditis in the first 48 hours following a transmural MI is common (c. 10% of patients). The pain is typical for pericarditis (worse on lying flat etc), a pericardial rub may be heard and a pericardial effusion may be demonstrated with an echocardiogram.

Dressler's syndrome tends to occur around 2-6 weeks following a MI. The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers. It is characterised by a combination of fever, pleuritic pain, pericardial effusion and a raised ESR. It is treated with NSAIDs.

Left ventricular aneurysm

The ischaemic damage sustained may weaken the myocardium resulting in aneurysm formation. This is typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated.



Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent pericardiocentesis and thoracotomy are required.

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. Acute hypotension and pulmonary oedema may occur. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 36 👎 22

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Suggest link

Report broken link

Score: **22.1%**

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A 43-year-old gentleman who is admitted and found to have a STEMI has chest pain on the ward. He is currently recovering from his percutaneous coronary intervention the day before and has felt well until now. The pain is over the left side of the chest and severe if he breathes. He has no cough or fevers and his observations are within normal limits.

On examination, he has normal heart sounds and a clear chest. He is mildly tender over the chest wall. His femoral puncture sites are clean with no haematoma. He is in sinus rhythm on the cardiac monitor and has soft non-oedematous calves. No JVP is seen.

Na^+	156 mmol/l
K^+	3.4 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
CRP	45 mg/l

Hb	134 g/l
Platelets	$398 * 10^9/l$
WBC	$13.0 * 10^9/l$

ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest x-ray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

- ○ Hospital acquired pneumonia ×
- ○ Pericarditis ×
- ○ Pulmonary embolism ×

○ Coronary artery dissection ×

○ Pleurisy ×

Submit answer

Reference ranges ⌵

Score: **22.1%**

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ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest x-ray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

Hospital acquired pneumonia	1%
Pericarditis	56%
Pulmonary embolism	6%

Coronary artery dissection	10%
Pleurisy	27%

Pleuritic chest pain at <48hrs after MI -> pericarditis

Important for me Less important

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospital-acquired pneumonia would usually take longer to develop. A coronary artery dissection can be a complication of PCI but there would most likely be ischaemic changes seen on the ECG. Pleurisy is usually a post inflammatory condition after a lower respiratory tract infection. Pulmonary embolism is possible but patients with MI are anticoagulated and this is early for a PE to develop. It is recognised that a number of MI patients develop and acute pericarditis in the first 48hrs. Treatment is supportive and it is key to rule out other complications first.

👍 👎 Discuss (12) Improve

Next question >

Myocardial infarction: complications ★

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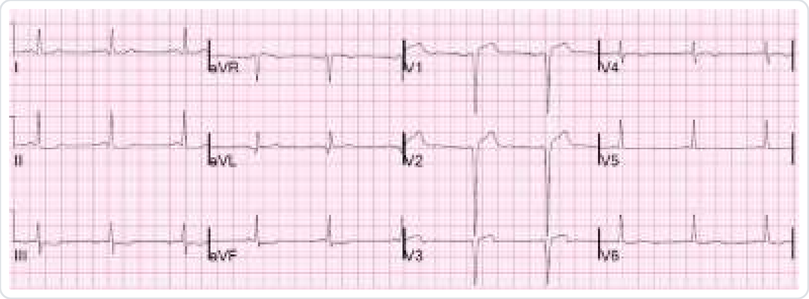

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+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 36 👎 22

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Suggest link Report broken link

Score: **21.8%**

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Question 88 of 129

A 63-year-old female patient attends the Emergency Department with crushing central chest pain and 3mm ST segment elevation in leads II, III and aVF. She is taken to the cardiac catheter laboratory where she has a primary PCI with a satisfactory angiographic result.

Six hours later, whilst on CCU, she develops complete heart block. The patient is asymptomatic and her haemodynamic parameters are as follows:

Pulse 44bpm, regular
Blood pressure - 123/75mmHg

What is the most appropriate course of action?

- ○ Synchronised direct current cardioversion (DCCV)
- ○ Continue close monitoring and observation of the patient
- ○ Start an infusion of isoprenaline
- ○ Insertion of a permanent pacemaker
- ○ Insertion of a temporary pacing wire

Submit answer

Reference ranges

Score: **21.8%**

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Pulse 44bpm, regular
Blood pressure - 123/75mmHg

What is the most appropriate course of action?

Synchronised direct current cardioversion (DCCV)	1%
Continue close monitoring and observation of the patient	74%
Start an infusion of isoprenaline	3%
Insertion of a permanent pacemaker	8%
Insertion of a temporary pacing wire	15%

In the description of the patient's presentation, it is stated that her ECG showed ST elevation in leads II, III and aVf, indicating an inferior STEMI. Complete heart block soon after an inferior MI is not uncommon, and usually resolves without the need for intervention. The key to this question is the fact that the patient is asymptomatic and haemodynamically stable. In view of this fact, it is sensible to closely monitor and observe on the assumption that given enough time post-reperfusion she will return to sinus rhythm. If she was haemodynamically unstable, a temporary pacing wire would be the best option in the first instance, with an upgrade to a permanent system if she did not recover to sinus rhythm in due course.

👍 👎 Discuss (4) Improve

Next question >

Complete heart block ★

Features

- syncope
- heart failure
- regular bradycardia (30-50 bpm)
- wide pulse pressure
- JVP: cannon waves in neck
- variable intensity of S1

Types of heart block

First degree heart block

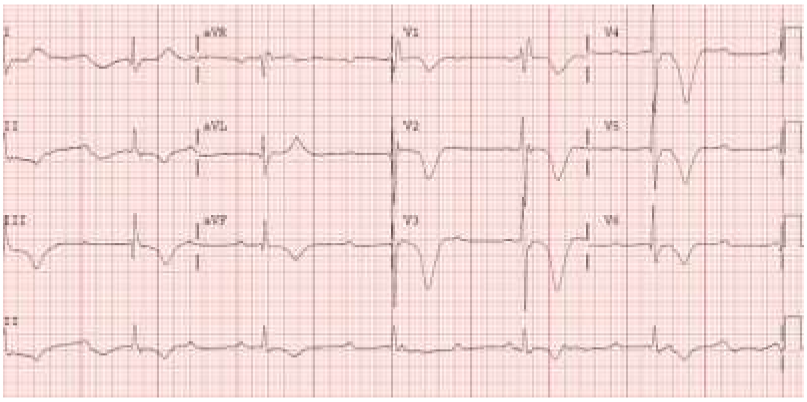
- PR interval > 0.2 seconds

Second degree heart block

- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex

Third degree (complete) heart block

- there is no association between the P waves and QRS complexes



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ECG showing third degree (complete) heart block

AI + Q 123

Next question >

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Extended textbook

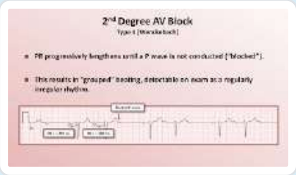
Links

ECGPedia 👍 13 👎 10

AV conduction problems

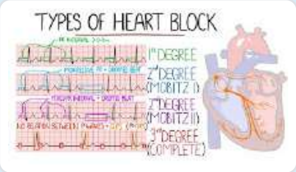
Suggest link Report broken link

Media


2nd Degree AV Block

AV block

Strong Medicine - YouTube 👍 2 👎 0


TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Atrioventricular block

Osmosis - YouTube 👍 0 👎 1

Report broken media

Score: **21.6%**

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A 28-year-old man presented with heart palpitations. He states that he has had a a couple of episodes each week for the past 3 months. He describes the palpitations as a rapid beating of his heart. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology. Inverted in lead I
PR interval	130ms
QRS	110ms. Loss of R wave progression in chest leads
QTc	410ms
Axis	Right axis deviation

What is the most likely explanation of the ECG results?

- ○ Torsades de pointes
- ○ Wolff-Parkinson-White (WPW) syndrome
- ○ Misplacement of the limb lead
- ○ Dextrocardia
- ○ AV nodal reentrant tachycardia (AVNRT)

Submit answer

Reference ranges

Score: **21.6%**

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Question 89 of 129 ✗

A 28-year-old man presented with heart palpitations. He states that he has had a a couple of episodes each week for the past 3 months. He describes the palpitations as a rapid beating of his heart. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology. Inverted in lead I
PR interval	130ms
QRS	110ms. Loss of R wave progression in chest leads
QTc	410ms
Axis	Right axis deviation

What is the most likely explanation of the ECG results?

Torsades de pointes	2%
Wolff-Parkinson-White (WPW) syndrome	20%
Misplacement of the limb lead	19%
Dextrocardia	39%
AV nodal reentrant tachycardia (AVNRT)	20%

Dextrocardia is associated with an inverted P wave in lead I, right axis deviation, and loss of R wave progression

Important for me Less important

The inverted P wave in lead I, right axis deviation, and loss of R wave progression should alert you to dextrocardia. Misplacement of the limb leads can cause a similar picture with inverted P wave in lead I and right axis deviation (RAD), however we would not expect loss of R wave progression, making dextrocardia the best answer in this question.

The PR interval is not short, and there is no delta waves present, making Wolff-Parkinson-White (WPW) unlikely.

The QTc is normal making Torsades de pointes unlikely.

Discuss (9) | Improve

Next question ›

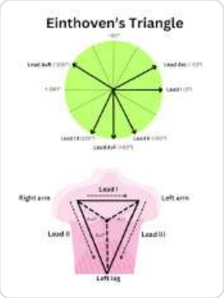
ECG: axis deviation

Causes of left axis deviation (LAD)

- left anterior hemiblock
- left bundle branch block
- inferior myocardial infarction
- Wolff-Parkinson-White syndrome* - right-sided accessory pathway
- hyperkalaemia
- congenital: ostium primum ASD, tricuspid atresia
- minor LAD in obese people

Causes of right axis deviation (RAD)

- right ventricular hypertrophy
- left posterior hemiblock
- lateral myocardial infarction
- chronic lung disease → cor pulmonale
- pulmonary embolism
- ostium secundum ASD
- Wolff-Parkinson-White syndrome* - left-sided accessory pathway
- normal in infant < 1 years old
- minor RAD in tall people



*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome

Next question >

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Links

Life in the Fastlane Left axis deviation	15	4
Life in the Fastlane ECG Axis Interpretation	16	4
Life in the Fastlane Right axis deviation	6	1

Suggest link

Report broken link

Score: **21.3%**

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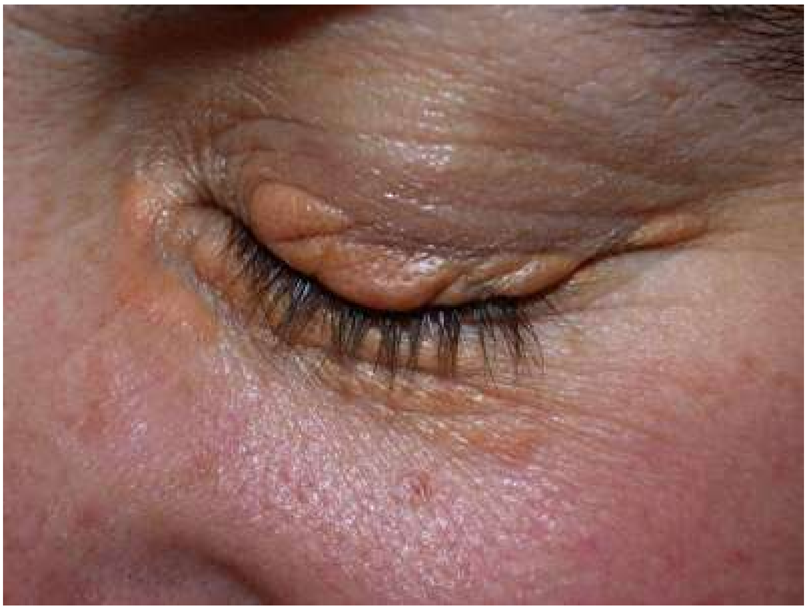
A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:



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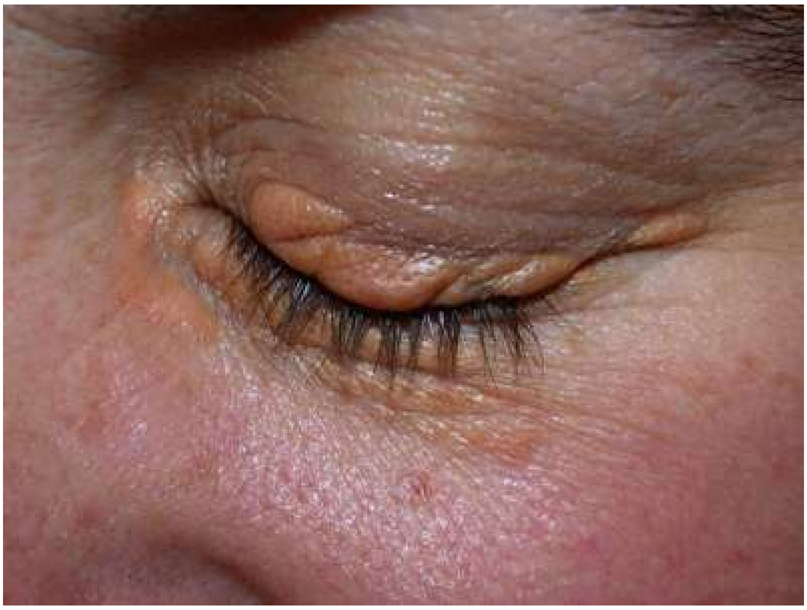
Given the likely diagnosis, which of the following would be an appropriate management option?

- ○ Topical emollient
- ○ Topical hydrocortisone
- ○ Topical ketoconazole
- ○ Topical trichloroacetic acid
- ○ Urgent referral to secondary care

Submit answer

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:



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Given the likely diagnosis, which of the following would be an appropriate management option?

Topical emollient	5%
Topical hydrocortisone	4%
Topical ketoconazole	3%
Topical trichloroacetic acid	45%
Urgent referral to secondary care	43%

The photo above demonstrates xanthelasma - high lipid levels leading to soft yellow/orange plaques, periorbitally. They are not of clinical concern

- except for the underlying lipid profile which should be investigated and treated accordingly. They can be left alone, but if patients are keen for treatment, a commonly used option is topical trichloroacetic acid.

Topical emollient is unnecessary. This will not help to alleviate the appearance of the xanthelasma.

Topical hydrocortisone is inappropriate here. As a steroid, this would not help to reduce the appearance of xanthelasma.

Topical ketoconazole is also inappropriate. This is an antifungal and may be used for seborrhoeic dermatitis, which would instead present with dry, flaky skin and erythema - not yellow/orange plaques.

An urgent referral to a hospital is also unwarranted. Xanthelasma is of no clinical concern and requires no further investigation.

👍 👎 Discuss (3) Improve

Next question >

Hyperlipidaemia: xanthomata ★

Characteristic xanthomata seen in hyperlipidaemia:

Palmar xanthoma
- remnant hyperlipidaemia
- may less commonly be seen in familial hypercholesterolaemia

Eruptive xanthoma are due to high triglyceride levels and present as multiple red/yellow vesicles on the extensor surfaces (e.g. elbows, knees)

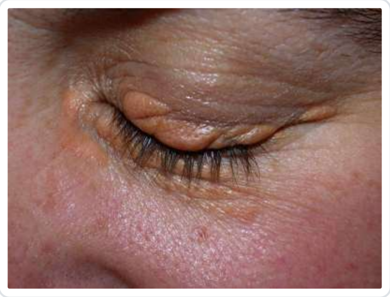
Causes of eruptive xanthoma
- familial hypertriglyceridaemia
- lipoprotein lipase deficiency

Tendon xanthoma, tuberous xanthoma, xanthelasma
- familial hypercholesterolaemia
- remnant hyperlipidaemia

Xanthelasma

Xanthelasma are yellowish papules and plaques caused by localized accumulation of lipid deposits commonly seen on the eyelid. They are also seen in patients without lipid abnormalities.

Management of xanthelasma, options include:
- surgical excision
- topical trichloroacetic acid
- laser therapy
- electrodesiccation



+ Q 123

Next question >

Textbooks

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Links

DermNet NZ 👍 12 👎 6

Xanthomas

Suggest link Report broken link

Score: **21.1%**

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Question 91 of 129

A 32-year-old woman presented to the emergency department with a 2-hour history of acute shortness of breath. Helical CT pulmonary angiography shows no evidence of pulmonary embolus but reveals dilatation of her ascending aorta to 4.4 cm.

On examination, she is afebrile and has a heart rate of 102 beats/min, respiratory rate of 19 breaths/min, and a BP of 132/46mmHg. Her cardiac examination reveals a normal S1 with an end-diastolic murmur over the aortic valve.

Which of the following physical examination findings are most likely to be present in this patient?

- ○ De musset's sign
- ○ Presence of atrial fibrillation
- ○ Pulsus bigeminus
- ○ Pulsus paradoxus
- ○ Pulsus parvus et tardus

Submit answer

Reference ranges

Score: **21.1%**

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Question 91 of 129 ✗

A 32-year-old woman presented to the emergency department with a 2-hour history of acute shortness of breath. Helical CT pulmonary angiography shows no evidence of pulmonary embolus but reveals dilatation of her ascending aorta to 4.4 cm.

On examination, she is afebrile and has a heart rate of 102 beats/min, respiratory rate of 19 breaths/min, and a BP of 132/46mmHg. Her cardiac examination reveals a normal S1 with an end-diastolic murmur over the aortic valve.

Which of the following physical examination findings are most likely to be present in this patient?

De musset's sign	66%
Presence of atrial fibrillation	2%
Pulsus bigeminus	9%
Pulsus paradoxus	13%
Pulsus parvus et tardus	10%

De Musset's sign (head bobbing) is a clinical sign of aortic regurgitation

Important for me Less important

The typical presentation of severe acute aortic regurgitation includes sudden, severe shortness of breath; rapid development of heart failure and chest pain. Heart sounds usually reveal a decrescendo diastolic murmur over the aortic area. The likely diagnosis here is severe aortic regurgitation as the patient has sudden shortness of breath with an end-diastolic murmur and wide pulse pressure and the dilatation of the aorta in CT angiography.
Wide pulse pressure is due to an incompetent aortic valve that allows the diastolic pressure to fall significantly often below 60mmHg.

Some of the important aortic regurgitation clinical signs are-

- Corrigan pulse - A 'water hammer' or 'collapsing' pulse is characterised by a rapidly rising and falling arterial pulse during late systole and diastole with wide pulse pressure. This finding is best appreciated by palpation of the radial or brachial arteries (exaggerated by raising the arm) or the carotid pulses.
- De Musset's sign - A head bob occurring with each heartbeat.
- Traube's sign - A pistol shot pulse (systolic and diastolic sounds) heard over the femoral arteries.
- Duroziez's sign - A systolic and diastolic bruit heard when the femoral artery is partially compressed.
- Quincke's pulses - capillary pulsations in the fingertips or lips.
- Mueller's sign - systolic pulsations of the uvula.

Pulsus paradoxus is described as a drop in systolic blood pressure of >10mmHg during the inspiratory phase, seen mostly in pericardial tamponade or severe COPD.

Pulses bigeminus occur in premature ventricular beats following a low volume pulse after a normal beat.

Pulsus parvus et tardus is more commonly known as a slow-rising pulse is a sign where on palpation of the pulse, it is late (tardus) relative to contraction of the heart and weak/small (parvus), found classically in severe aortic stenosis.

The presence of atrial fibrillation is usually present in enlarged left atrial like severe mitral stenosis or mitral regurgitation.

👍 👎 Discuss (8) Improve

Next question ›

Aortic regurgitation ★

Aortic regurgitation (AR) is the leaking of the aortic valve of the heart that causes blood to flow in the reverse direction during ventricular diastole.

It can be caused either by disease of the aortic valve or by distortion or dilation of the aortic root and ascending aorta.

	Causes of AR due to valve disease	Causes of AR due to aortic root disease
Chronic presentation	- rheumatic fever: the most common cause in the developing world - calcific valve disease - connective tissue diseases e.g. rheumatoid arthritis/SLE - bicuspid aortic valve (affects both the valves and the aortic root)	- bicuspid aortic valve (affects both the valves and the aortic root) - spondylarthropathies (e.g. ankylosing spondylitis) - hypertension - syphilis - Marfan's, Ehler-Danlos syndrome
Acute presentation	- infective endocarditis	- aortic dissection

Features
- early diastolic murmur: intensity of the murmur is increased by the handgrip manoeuvre
- collapsing pulse
- wide pulse pressure
- Quincke's sign (nailbed pulsation)
- De Musset's sign (head bobbing)
- mid-diastolic Austin-Flint murmur in severe AR - due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams

Suspected AR should be investigated with echocardiography.

Management
- medical management of any associated heart failure
- surgery: aortic valve indications include

  - symptomatic patients with severe AR
  - asymptomatic patients with severe AR who have LV systolic dysfunction

+ Q 123

Next question >

Textbooks

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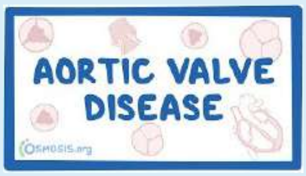
Media

Aortic regurgitation murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 0 👎 0



Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 6 👎 1

Report broken media

Score: **20.9%**

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← Question 92 of 129 →

A 77-year-old man with known atrial fibrillation is admitted following an upper gastrointestinal haemorrhage. His atrial fibrillation is managed using bisoprolol and warfarin. Since his admission, he has had four large episodes of haematemesis. You, the emergency department doctor, request the patient's INR to be checked as one of a series of investigations. The haematology laboratory phone through and inform you his INR is 8.5. He is currently hypotensive (90/45 mmHg) and tachycardic (120 beats per minute). You begin resuscitation using 0.9% saline, and send a cross match, group and save. What is the most appropriate treatment of this patients INR?

- ○ Fresh frozen plasma + stop warfarin ×
- ○ Vitamin K + stop warfarin ×
- ○ Prothrombin complex concentrates ×
- ○ Prothrombin complex concentrates + vitamin K + stop warfarin ×
- ○ Stop warfarin ×

Submit answer

Reference ranges

Score: **20.9%**

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Fresh frozen plasma + stop warfarin	2%
Vitamin K + stop warfarin	4%
Prothrombin complex concentrates	3%
Prothrombin complex concentrates + vitamin K + stop warfarin	91%
Stop warfarin	0%

Major bleeding - stop warfarin, give intravenous vitamin K 5mg, prothrombin complex concentrate

Important for me Less important

The nub of this question is the emergency management of haemorrhage in patients on warfarin. This patient has an INR greater than 8 and is actively bleeding. Therefore the answer is 4.

Patients on warfarin have reduced levels of Factor X, IX, VII and II. Rapid correction is most effectively achieved through administration of prothrombin complex concentrates.

The British Journal of Haematology states that: 'Emergency anticoagulation reversal in patients with major bleeding should be with 2550 u/kg four-factor prothrombin complex concentrate and 5 mg intravenous vitamin K'

Follow this link for more information
http://www.bcshguidelines.com/documents/warfarin4thed.pdf

Discuss (3) Improve

Next question

Warfarin: management of high INR

The following is based on the BNF guidelines, which in turn take into account the British Committee for Standards in Haematology (BCSH) guidelines.

Situation	Management
Major bleeding (e.g. variceal haemorrhage, intracranial haemorrhage)	Stop warfarin Give intravenous vitamin K 5mg Prothrombin complex concentrate - if not available then FFP*
INR > 8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Repeat dose of vitamin K if INR still too high after 24 hours Restart warfarin when INR < 5.0
INR > 8.0 **No bleeding**	Stop warfarin Give vitamin K 1-5mg by mouth, using the intravenous preparation orally Repeat dose of vitamin K if INR still too high after 24 hours Restart when INR < 5.0
INR 5.0-8.0 **Minor bleeding**	Stop warfarin Give intravenous vitamin K 1-3mg Restart when INR < 5.0

Situation	Management
INR 5.0-8.0 **No bleeding**	Withhold 1 or 2 doses of warfarin Reduce subsequent maintenance dose

*as FFP can take time to defrost prothrombin complex concentrate should be considered in cases of intracranial haemorrhage

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF Warfarin	👍 16 👎 11
BNF Oral anticoagulants	👍 18 👎 10

Suggest link

Report broken link

Score: **21.7%**

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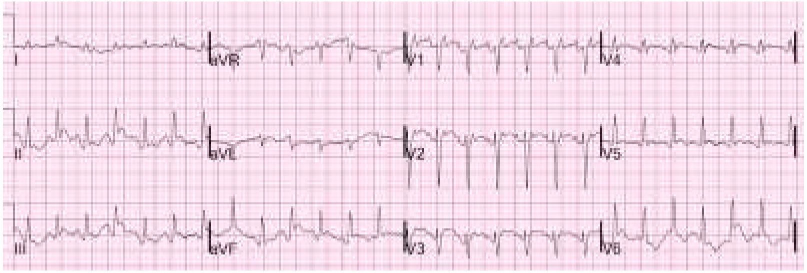
A 70-year-old man is admitted with fever, chills and dysuria. He has a medical history of ischaemic heart disease, hypertension, hyperlipidaemia and diabetes. His current medications include aspirin, ramipril, bisoprolol, atorvastatin, metformin and dapagliflozin. He is an ex-smoker and requires assistance with personal care. He is started on cefuroxime for urosepsis. His admission ECG shows:



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What is the most likely diagnosis?

○ Atrial fibrillation

○ Atrial flutter

○ Sinus tachycardia

○ Ventricular tachycardia

○ Wolff Parkinson White syndrome

Submit answer

Reference ranges

Score: **21.7%**

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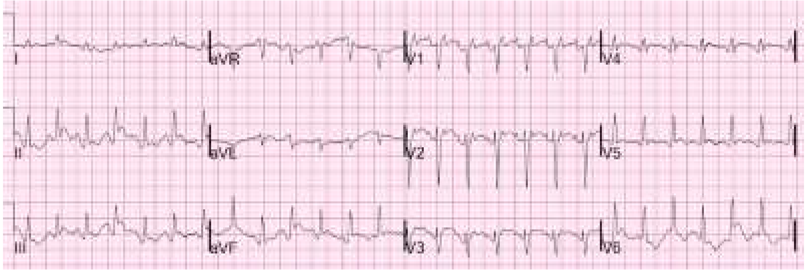
Question 93 of 129 ×

A 70-year-old man is admitted with fever, chills and dysuria. He has a medical history of ischaemic heart disease, hypertension, hyperlipidaemia and diabetes. His current medications include aspirin, ramipril, bisoprolol, atorvastatin, metformin and dapagliflozin. He is an ex-smoker and requires assistance with personal care. He is started on cefuroxime for urosepsis. His admission ECG shows:



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What is the most likely diagnosis?

Atrial fibrillation	12%
Atrial flutter	45%
Sinus tachycardia	27%
Ventricular tachycardia	4%
Wolff Parkinson White syndrome	12%

The ECG shows an atrial rate of 300 bpm and ventricular rate of 150 bpm suggesting atrial flutter with a 2:1 AV block (regular p waves seen as notched t waves can be better appreciated in lead III, V1 and V2).

Atrial flutter is the correct answer. There are regular saw-toothed P waves at 300 bpm. There are regular QRS complexes at 150 bpm. Thus, this is atrial flutter with a rapid ventricular response and 2:1 AV block.

Atrial fibrillation is incorrect. Atrial fibrillation, unlike atrial flutter, is irregular with fibrillatory p waves caused by disorganised and chaotic atrial electrical activity. Contrary to atrial fibrillation, atrial flutter is more amenable to cardioversion than medications.

Sinus tachycardia is incorrect. In sinus tachycardia, every P wave is followed by a QRS complex and the PR interval is <200 ms. However, in this patient's ECG, not every p wave is followed by a QRS complex suggesting a 2:1 AV block. Moreover, the saw-toothed P waves at 300 bpm and prolonged PR interval is suggestive of atrial flutter.

Ventricular tachycardia is a broad complex tachycardia. The QRS complex here is narrow measuring < 120 ms and regular at 150 bpm suggesting an Atrial Flutter with 2:1 block.

Wolff Parkinson White syndrome is incorrect. WPW is a pre-excitation syndrome characterised by PR interval <120 ms, delta wave and QRS >110 ms which are absent in this patient's ECG.

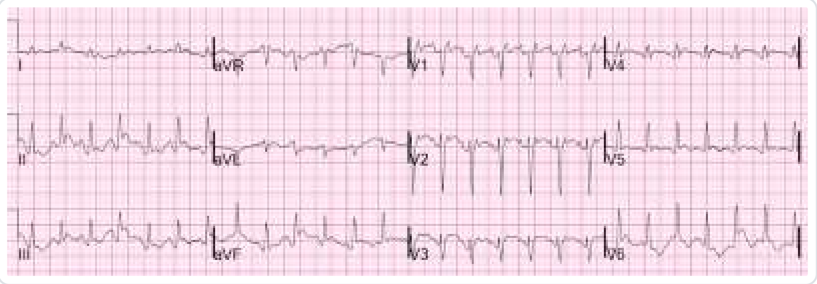
👍 👎 Discuss (1) Improve

Next question >

Atrial flutter ★

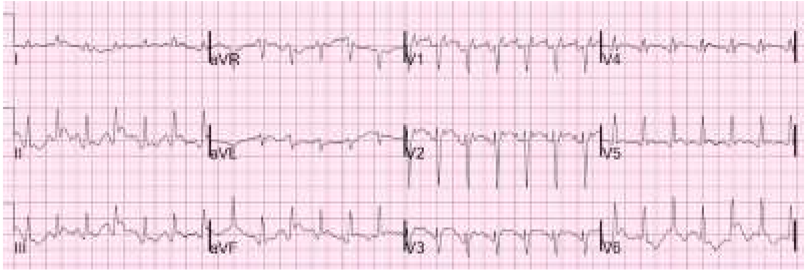
Atrial flutter is a form of supraventricular tachycardia characterised by a succession of rapid atrial depolarisation waves.

ECG findings
- 'sawtooth' appearance
- as the underlying atrial rate is often around 300/min the ventricular or heart rate is dependent on the degree of AV block. For example if there is 2:1 block the ventricular rate will be 150/min
- flutter waves may be visible following carotid sinus massage or adenosine



Management

- is similar to that of atrial fibrillation although medication may be less effective
- atrial flutter is more sensitive to cardioversion however so lower energy levels may be used
- radiofrequency ablation of the tricuspid valve isthmus is curative for most patients



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123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 19 9

Atrial Flutter

Suggest link

Report broken link

Media

Atrial flutter (type 1, type 2)

Osmosis - YouTube 👍 3 👎 0

Report broken media

Score: **21.5%**

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A 77 year old woman is brought to the Emergency Department by blue light ambulance in acute respiratory distress. She has a background history of congestive cardiac failure (NYHA II) due to hypertension and a previous anterior myocardial infarction which was treated medically. She is also a type 2 diabetic and has chronic kidney disease stage 2. She takes aspirin 75mg od, simvastatin 40mg od, ramipril 5mg od, bisoprolol 5mg od, furosemide 40mg bd and vildagliptin 5mg od.

On assessment her airway is patent. Respiratory rate is 32 per minute. Fingertip oxygen saturations are 90% on 15L/min oxygen. There are bilaterally crackles in the chest to above the midzones. Heart rate is 112bpm. Blood pressure is 125/75mmHg. ECG shows sinus tachycardia with no acute ischaemic change. There is peripheral oedema to the mid thigh and the JVP is elevated 6cm.

Which medical therapy is most appropriate immediately?

- ○ 3mg bumetanide orally
- ○ 80mg furosemide intravenously
- ○ 300mg aspirin, 300mg clopidogrel and 2.5mg subcutaneous fondaparinux
- ○ 50mg glycerol trinitrate infusion, commencing at 1mg/hr
- ○ 5mg metoprolol intravenously

Submit answer

Reference ranges

Score: **21.5%**

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Question 94 of 129 ✗

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Which medical therapy is most appropriate immediately?

3mg bumetanide orally	1%
80mg furosemide intravenously	93%
300mg aspirin, 300mg clopidogrel and 2.5mg subcutaneous fondaparinux	1%
50mg glycerol trinitrate infusion, commencing at 1mg/hr	4%
5mg metoprolol intravenously	0%

IV loop diuretic is the treatment for acute pulmonary oedema

Important for me Less important

This question concerns the pharmacological management of acute decompensation of left ventricular failure. It is important to point out that a patient presenting with symptoms of acute heart failure does not automatically imply an acute ischaemic event. Cardiac failure can be precipitated by changes in medication, intercurrent illness, worsening of other medical conditions or even dietary changes and it is wrong to assume a patient presenting with symptoms of cardiac failure have an acute coronary syndrome de facto. These patients should not routinely be given anticoagulant triple therapy unless there is also evidence of coronary ischaemic disease since this is unnecessary and significantly increases risk of haemorrhage. Prophylaxis against thromboembolic disease in cardiac failure is important however.

Current guidelines issued by the European Society of Cardiology and echoed by NICE guidance in the UK recommend diuretic therapy as first line medical intervention in acute cardiac failure. Intravenous administration is suggested rather than oral since this route is more rapid, there is a higher bioavailability and gut absorption can be hampered by gastrointestinal oedema in severe heart failure. Even pending the results of renal function, if pulmonary oedema is so severe to be impeding gas exchange, a bolus of furosemide is recommended to commence offloading. In patients with pre-existing renal disease, higher doses of diuretics will be needed to achieve adequate diuresis. Renal disease in itself it not a reason to avoid diuretics in heart failure, however diuretic use will result in a deterioration in renal function. Diuretic use will cause a rapid depletion in intravascular volume but the aim is to reduce tissue fluid by reducing hydrostatic pressure and a balance must be struck between deteriorating renal function due to intravascular loss and failure of therapy. Careful fluid balance monitoring is required.

Other options for fluid offloading include positive pressure ventilation which has a good evidence base behind it but patients find it uncomfortable and claustrophobic. Pressure ventilation causes an increase in intrathoracic pressure so venous return to the heart is reduced which in turn reduces cardiac output; care must be taken in hypotensive patients. Similarly glycerol trinitrate (GTN) can be used to offload pulmonary fluid by dilating arteries and to a lesser extent veins. This reduces both afterload and preload respectively reducing pulmonary oedema. Evidence demonstrates that this effect is negligible however when compared to standard diuretic therapy and should not be used first line. Indeed, it is usually only recommended in hypertensive patients with heart failure; neither GTN nor opioids are considered appropriate initial treatment of heart failure any longer, despite ongoing ubiquitous use. Side effects of GTN include hypotension, rapid tolerance, headache and complete cardiovascular collapse in cardiac outflow tract obstructions; GTN should never be given to patients with severe aortic stenosis for this reason.

Beta-blockers are useful in stable heart failure since they reduce myocardial workload and hence oxygen demand, improve diastolic filling by slowing the rate down and have been proven to improve outcomes. Their use in acute heart failure is less certain and intravenous metoprolol can precipitate cardiovascular collapse due to hypotension, severe bradycardia or dyssynchrony. Sinus tachycardia or atrial fibrillation in heart failure is best treated with intravenous digoxin, amiodarone or DC cardioversion.

👍 👎 Discuss (6) Improve

Next question >

Heart failure: acute management

Recommended treatments for all patients
- IV loop diuretics
    - e.g. furosemide or bumetanide

Possible additional treatments
- oxygen
    - this should be given in line with British Thoracic Society guidelines, i.e. keep oxygen saturations at 94-98%
- vasodilators
    - nitrates should not be routinely given to all patients
    - they may, however, have a role if concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease
    - the major side-effect/contraindication is hypotension

Patients with respiratory failure
- CPAP

Patients with hypotension (e.g. < 85 mmHg)/cardiogenic shock

- this can be a difficult scenario to manage
    - some of the treatments typically used for acute heart failure (e.g. loop diuretics and nitrates) may exacerbate the hypotension
- inotropic agents
    - e.g. dobutamine
    - should be considered for patients with severe left ventricular dysfunction who have potentially reversible cardiogenic shock
- vasopressor agents
    - e.g. norepinephrine
    - normally only used if insufficient response to inotropes and evidence of end-organ hypoperfusion
- mechanical circulatory assistance
    - e.g. intra-aortic balloon counterpulsation or ventricular assist devices

Other treatments

- opiates
    - NICE state 'do not routinely offer opiates to people with acute heart failure'
    - they were previously used routinely and help to reduce dyspnoea/distress in patients
    - however some studies have suggested increased morbidity in patients given opiates

General points

- regular medication for heart failure such as beta-blockers ACE-inhibitors should be continued
    - beta-blockers should only be stopped if the patient has heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

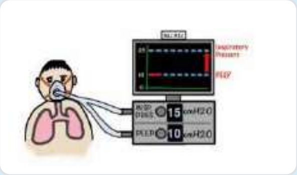
Links

NICE 👍 6 👎 9

2014 Acute heart failure: diagnosis and management

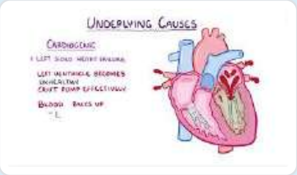
Suggest link Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 👍 17 👎 0


UNDERLYING CAUSES
CARDIOGENIC

Pulmonary oedema

Osmosis - YouTube 👍 4 👎 0

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Score: **21.3%**

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93	✗
94	✗

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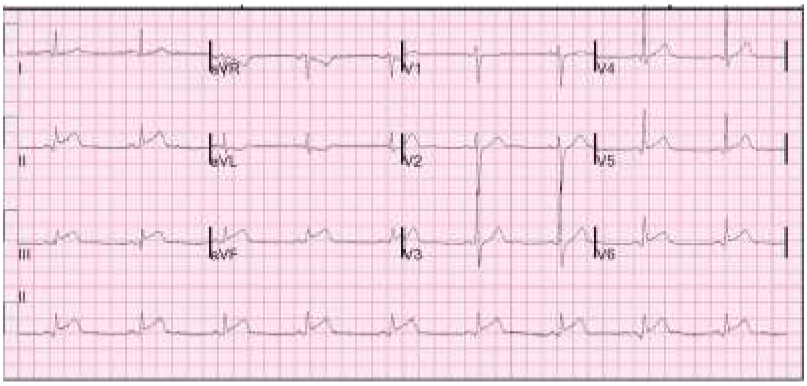
Question 95 of 129

A 64-year-old man presents to the emergency department with a 2-hour history of chest pain. He has never experienced pain like this before. There is no history of shortness of breath and he reports to have been well in himself prior to today. His past medical history includes hypertension and hyperlipidaemia.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

● Anterior myocardial infarction

○ Anterolateral myocardial infarction

○ Inferior myocardial infarction

○ Inferolateral myocardial infarction

○ Septal myocardial infarction

Submit answer

Reference ranges

Question 95 of 129 ✗

A 64-year-old man presents to the emergency department with a 2-hour history of chest pain. He has never experienced pain like this before. There is no history of shortness of breath and he reports to have been well in himself prior to today. His past medical history includes hypertension and hyperlipidaemia.

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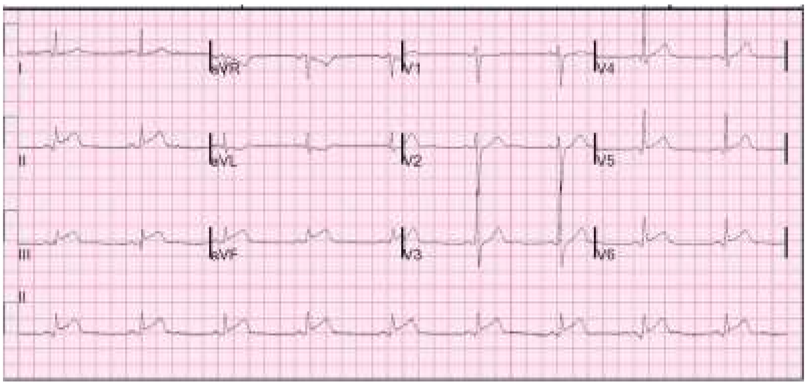

© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Anterior myocardial infarction	1%
Anterolateral myocardial infarction	2%
Inferior myocardial infarction	48%
Inferolateral myocardial infarction	49%
Septal myocardial infarction	0%

Inferolateral myocardial infarction is correct. This patient's ECG shows ST elevation in leads II, III, aVF, V5 and V6 along with ST depression in aVL. This is indicative of an inferolateral myocardial infarction. The inferior wall of the myocardium is supplied by both the right coronary artery (medial inferior wall) and left circumflex artery (lateral inferior wall). Given the ST elevation in the inferior and lateral leads, the left circumflex artery is more likely to be the occluded vessel.

Anterior myocardial infarction is incorrect. This presents with ST elevation in leads V1-V2 (septal) and V3-V4 (anterior). This is not seen here.

Anterolateral myocardial infarction is incorrect. This presents with ST elevation in the anterior leads (V3 and V4) +/- the septal leads (V1 and V2) in addition to ST elevation in the lateral leads (I, aVL, V5 and V6). Although there is ST elevation in V5 and V6, there are no ST segment changes in the anterior leads.

Inferior myocardial infarction is incorrect. Although there is ST elevation in the inferior leads (II, III and aVF), there is also ST elevation in V5 and V6 which makes an inferolateral myocardial infarction the diagnosis.

Septal myocardial infarction is incorrect. A septal myocardial infarction presents with ST elevation in leads V1 and V2 which is not seen here.

Discuss (8) | Improve

Next question >

ECG: coronary territories ★

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex

	ECG changes	**Coronary artery**
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

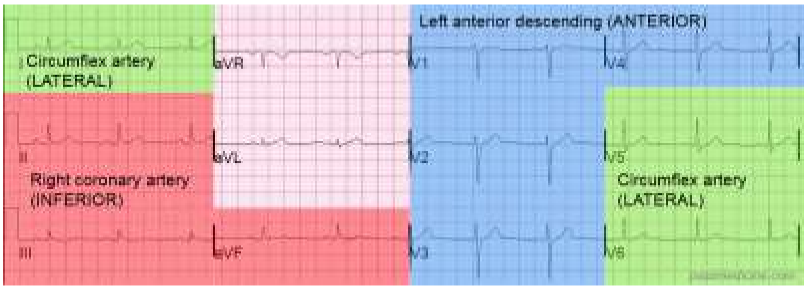
Left anterior descending (ANTERIOR)
Circumflex artery (LATERAL)
aVR
V1
V4
II
aVL
V2
V5
Right coronary artery (INFERIOR)
Circumflex artery (LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

Next question

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal 👍 7 👎 11

ECG correlation with coronary anatomy

University of Massachusetts Medical School 👍 2 👎 6

Classic circumflex infarction ECG

Suggest link Report broken link

Score: **21.1%**

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An 82-year-old lady presented with shortness of breath and palpitations. These came on suddenly whilst she was watching television. She had a sedentary lifestyle and recently had been confined to bed with an upper respiratory tract infection. After this she had noticed that her ankles and legs were more swollen than usual, in particular her left leg. On further questioning she said that she had always needed three pillows to sleep but had recently required an extra pillow.

Her past medical history included hypertension, hypothyroidism, asthma and known coronary artery disease.

On examination her pulse was irregularly irregular. The jugular venous pressure was 6cm above the manubriosternal angle. Heart sounds were normal with no added sounds. On auscultation of the lung fields there were coarse crepitations at both bases. There was bilateral pitting oedema to the knees more marked on the left. The circumference of the left leg was 2cm greater than the right.

Observations:

- Heart rate: 128 beats per minute
- SaO2: 92% on room air
- Respiratory rate: 22 breaths per minute
- Temperature: 36.8 degrees Celsius
- Blood pressure: 110/68 mmHg

Investigations:

- 12-lead electrocardiogram: atrial fibrillation with a fast ventricular response, left ventricular hypertrophy
- Chest X-ray: cardiomegaly, Kerley B lines, small bilateral pleural effusions, pulmonary oedema
- Echocardiogram: poor biventricular function with an ejection fraction of 35%
- Computed Tomography Pulmonary Angiogram: small segmental pulmonary embolus

This lady was anticoagulated appropriately. Following this acute episode she improved clinically but remained in atrial fibrillation.

Given the clinical information available which of the following is the most appropriate drug to control her heart rate in the long term?

- ○ Metoprolol ×
- ○ Digoxin ×
- ○ Diltiazem ×
- ○ Flecainide ×
- ○ Warfarin ×

Submit answer

Reference ranges ∨

Score: **21.1%**

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Question 96 of 129 ✕

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This lady was anticoagulated appropriately. Following this acute episode she improved clinically but remained in atrial fibrillation.

Given the clinical information available which of the following is the most appropriate drug to control her heart rate in the long term?

🟥 Metoprolol	13%
🟩 Digoxin	70%
Diltiazem	13%
Flecainide	3%
Warfarin	2%

Digoxin is a good choice as this patient also has evidence of congestive cardiac failure which may also benefit from digoxin therapy. It is important to note that NICE recommends the use of digoxin only in sedentary patients and not those with an active lifestyle.

Metoprolol, a beta blocker, is not an appropriate choice here given the past medical history of asthma.

Warfarin is not a drug that is used for rate control; it is an anticoagulant.

Diltiazem is a rate-limiting calcium channel blocker and unlike digoxin does not have the added benefits in congestive cardiac failure.

Flecainide, a class 1c anti-arrhythmic drug, is used for chemical cardioversion in patients with no underlying structural heart disease as part of a pill-in-the-pocket regimen.

For further information on the treatment of atrial fibrillation see NICE guidance: http://egap.evidence.nhs.uk/atrial-fibrillation-cg36/guidance

👍 | 👎 | 💬 Discuss (11) | Improve

Next question >

Chronic heart failure: drug management ★

NICE issued updated guidelines on management in 2018, key points are summarised here.

Whilst loop diuretics play an important role in managing fluid overload it should be remembered that no long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide.

First-line therapy

The first-line treatment for all patients is both an **ACE-inhibitor** and a **beta-blocker**
- generally, one drug should be started at a time. NICE advise that clinical judgement is used when determining which one to start first
- beta-blockers licensed to treat heart failure in the UK include bisoprolol, carvedilol, and nebivolol.
- ACE-inhibitors and beta-blockers have no effect on mortality in heart failure with *preserved* ejection fraction

Second-line therapy

The standard second-line treatment is an **aldosterone antagonist**
- these are sometimes referred to as mineralocorticoid receptor antagonists. Examples include spironolactone and eplerenone
- it should be remembered that both ACE inhibitors (which the patient is likely to already be on) and aldosterone antagonists both cause hyperkalaemia - therefore potassium should be monitored

There is an increasing role for **SGLT-2 inhibitors** in the management of heart failure with a reduced ejection fraction
- these drugs reduce glucose reabsorption and increase urinary glucose excretion
- examples include canagliflozin, dapagliflozin and empagliflozin
- the evidence base shows SGLT-2 inhibitors reduced hospitalisation secondary to heart failure and cardiovascular death
- international guidelines widely recommend their usage. In terms of NICE, a technology appraisal from 2021 support the use of dapagliflozin as an add-on to optimised standard care

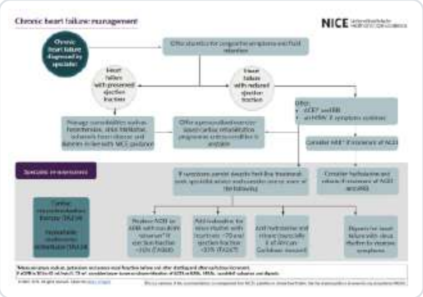
Third-line therapy

Third-line treatment should be initiated by a specialist. Options include ivabradine, sacubitril-valsartan, hydralazine in combination with nitrate, digoxin and cardiac resynchronisation therapy

- ivabradine
    - criteria: sinus rhythm > 75/min and a left ventricular fraction < 35%
- sacubitril-valsartan
    - criteria: left ventricular fraction < 35%
    - is considered in heart failure with reduced ejection fraction who are symptomatic on ACE inhibitors or ARBs
    - should be initiated following ACEi or ARB wash-out period
- digoxin
    - digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties
    - it is strongly indicated if there is coexistent atrial fibrillation
- hydralazine in combination with nitrate
    - this may be particularly indicated in Afro-Caribbean patients
- cardiac resynchronisation therapy
    - indications include a widened QRS (e.g. left bundle branch block) complex on ECG

Other treatments

- offer **annual influenza vaccine**
- offer one-off **pneumococcal vaccine**
    - adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years


Chronic heart failure management
NICE
Chronic heart failure diagnosed by specialist
Heart failure with preserved ejection fraction
Heart failure with reduced ejection fraction

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 14 👎 13

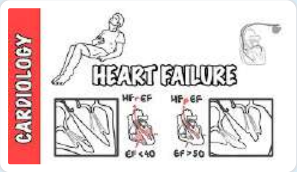
2012 Guidance on ivabradine

NICE 👍 42 👎 15

2018 Chronic heart failure guidelines

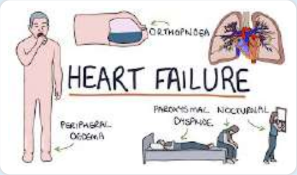
Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 1 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube 👍 1 👎 1

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 0 👎 2

Report broken media

Score: **20.8%**

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96 ✗

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Question 97 of 129

A 68-year-old lady, who is a retired teacher, is admitted with lightheadedness. She has a history relevant for depression, osteoarthritis and sciatica. Her medication history includes codeine 30mg QDS, ibuprofen 400mg TDS, pregabalin 50mg TDS and amitriptyline 100mg ON. She is also on nitrofurantoin 50mg OD for recurrent urinary tract infections. Electrocardiogram (ECG) confirms normal sinus rhythm with a heart rate of 80bpm. There are no dynamic ST/T changes. PR interval is 140ms and QTc is prolonged at 526ms.

Which medication is most likely to be implicated?

- ○ Nitrofurantoin ×
- ○ Pregabalin ×
- ○ Amitriptyline ×
- ○ Ibuprofen ×
- ○ Codeine ×

Submit answer

Reference ranges

Score: **20.8%**

1 ✗
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Question 97 of 129 ✓

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Which medication is most likely to be implicated?

Nitrofurantoin	10%
Pregabalin	2%
Amitriptyline	86%
Ibuprofen	0%
Codeine	2%

The correct answer is amitriptyline. Tricyclic anti-depressants are associated with prolongation of QTc interval. Other medications that can cause this include quinidine, erythromycin, digoxin, amiodarone and lithium. Prolongation of QTc interval, if marked, can result in R on T phenomenon, whereby a ventricular stimulus causes premature depolarisation of cells that have not completely repolarised. This may result in ventricular tachycardia or ventricular fibrillation. This lady is presenting with dizziness, and one should be concerned that this is a result of dysrhythmia secondary to pharmacotherapy. Any implicated medications should be temporarily discontinued, with alternatives sought.

Discuss (4) | Improve

Next question

Long QT syndrome ★

Long QT syndrome (LQTS) is an inherited condition associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia/torsade de pointes and can therefore cause collapse/sudden death. The most common variants of LQTS (LQT1 & LQT2) are caused by defects in the alpha subunit of the slow delayed rectifier potassium channel. A normal corrected QT interval is less than 430 ms in males and 450 ms in females.

Causes of a prolonged QT interval:

Congenital	**Drugs***	**Other**
• Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel) • Romano-Ward syndrome (no deafness)	• amiodarone, sotalol, class 1a antiarrhythmic drugs • tricyclic antidepressants, selective serotonin reuptake inhibitors (especially citalopram) • methadone • chloroquine • terfenadine** • erythromycin • haloperidol • ondanestron	• electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia • acute myocardial infarction • myocarditis • hypothermia • subarachnoid haemorrhage

Features

- may be picked up on routine ECG or following family screening
- Long QT1 - usually associated with exertional syncope, often swimming
- Long QT2 - often associated with syncope occurring following emotional stress, exercise or auditory stimuli
- Long QT3 - events often occur at night or at rest
- sudden cardiac death

Management

- avoid drugs which prolong the QT interval and other precipitants if appropriate (e.g. Strenuous exercise)
- beta-blockers***
- implantable cardioverter defibrillators in high risk cases

*the usual mechanism by which drugs prolong the QT interval is blockage of potassium channels. See the link for more details

**a non-sedating antihistamine and classic cause of prolonged QT in a patient, especially if also taking P450 enzyme inhibitor, e.g. Patient with a cold takes terfenadine and erythromycin at the same time

***note sotalol may exacerbate long QT syndrome

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Pharmacolgy Review 👍 13 👎 8

2010 Drug-Induced Long QT syndrome

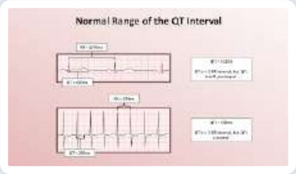
Life in the Fast Lane

👍 31 👎 9

QT Interval and Long QT syndrome

Suggest link Report broken link

Media


Normal Range of the QT Interval

Advanced EKGs - The QT Interval and Long QT Syndrome

Strong Medicine - YouTube 👍 7 👎 0

Long QT syndrome & torsade de pointes

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **21.6%**

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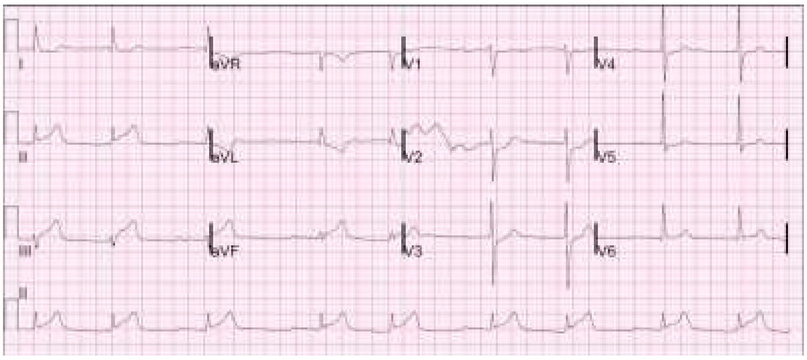
A 67-year-old man presents to the Emergency Department with central crushing chest pain. His ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

What is shown on the ECG?

- ○ Anterior non-ST elevation MI + atrioventricular block
- ○ Inferior ST elevation MI + atrioventricular block
- ○ Inferior ST elevation MI + left bundle branch block
- ○ Inferior ST elevation MI + right bundle branch block
- ○ Anterior non-ST elevation MI + complete heart block

Submit answer

Reference ranges

Score: **21.6%**

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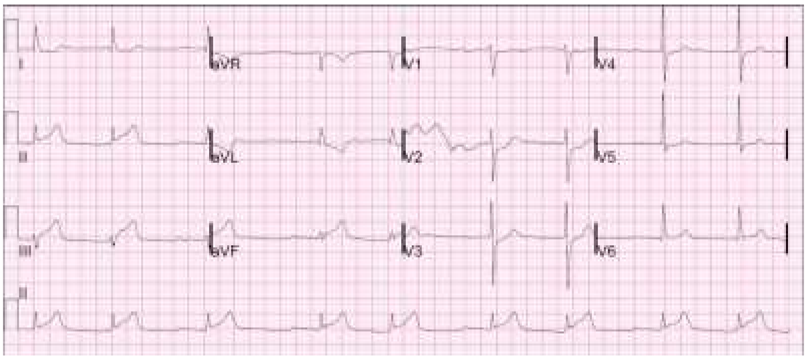
Question 98 of 129

A 67-year-old man presents to the Emergency Department with central crushing chest pain. His ECG is shown below:



© Image used on license from Dr Smith, University of Minnesota

What is shown on the ECG?

Anterior non-ST elevation MI + atrioventricular block	1%
Inferior ST elevation MI + atrioventricular block	83%
Inferior ST elevation MI + left bundle branch block	8%
Inferior ST elevation MI + right bundle branch block	5%
Anterior non-ST elevation MI + complete heart block	3%

There is ST elevation in the II, III and aVF diagnostic of an inferior myocardial infarction. The PR interval is significantly prolonged (first degree heart block) suggestive that the AV node may have been affected by the infarction.

Discuss (5) | Improve

Next question ›

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V1-6, I, aVL	Proximal left anterior descending
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Changes in V1-3 Reciprocal changes of STEMI are typically seen: • horizontal ST depression • tall, broad R waves • upright T waves • dominant R wave in V2 Posterior infarction is confirmed by ST elevation and Q waves in posterior leads (V7-9)	Usually left circumflex, also right coronary

It should be remembered that a new left bundle branch block (LBBB) may point towards a diagnosis of acute coronary syndrome.

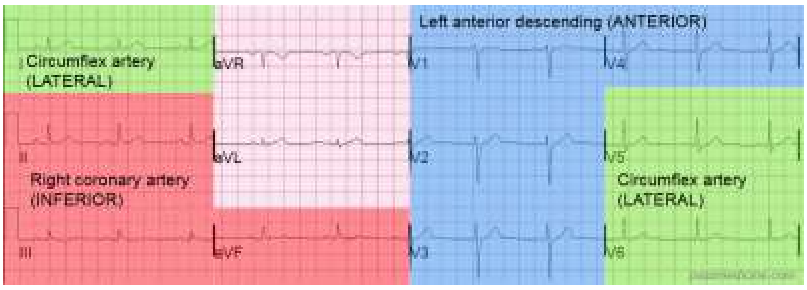

Circumflex artery
(LATERAL)
aVR
Left anterior descending (ANTERIOR)
V1
V4
II
Right coronary artery
(INFERIOR)
aVL
V2
V5
Circumflex artery
(LATERAL)
III
aVF
V3
V6

Diagram showing the correlation between ECG changes and coronary territories in acute coronary syndrome

123

Next question >

Posterior MI typically present on ECG with tall R waves V1-2

Textbooks

High-yield textbook

Extended textbook

Links

Postgraduate Medical Journal ECG correlation with coronary anatomy	👍	7	👎	11
University of Massachusetts Medical School Classic circumflex infarction ECG	👍	2	👎	6

Suggest link

Report broken link

Score: **21.4%**

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Back to top

A 54-year-old woman presents to the emergency department with shortness of breath. She has slight associated pleuritic chest pain, but no cough and denies coughing up any blood and denies feeling hot or cold. Her symptoms started four hours ago and came on suddenly whilst at rest. She was well prior to today.

She has a past medical history of hypothyroidism and underwent a wide local excision for ductal carcinoma in situ one year ago. Her observations are all within normal range except for oxygen saturations which are at 93%. She has never smoked. Chest examination reveals few scattered crepitations heard over the right lower zone. Blood tests, including a D-dimer, are pending. What is the most appropriate next step?

○ Warfarin ×

○ Chest X-ray ×

○ CT pulmonary angiogram ×

○ Ventilation-Perfusion scan ×

○ Treatment dose low molecular weight heparin ×

Submit answer

Reference ranges

Score: **21.4%**

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Warfarin	1%
Chest X-ray	59%
CT pulmonary angiogram	23%
Ventilation-Perfusion scan	1%
Treatment dose low molecular weight heparin	16%

A chest xray is an essential investigation when investigating a PE

Important for me Less important

The correct answer is chest X-ray. This is a patient presenting with a typical history of a pulmonary embolism (PE). Whilst a CT pulmonary angiogram should be arranged urgently, a chest X-ray should be done first to exclude alternative pathologies such as a chest infection or pneumothorax. If a chest X-ray was done and a definitive scan such as a CT pulmonary angiogram or ventilation-perfusion scan was not available immediately then starting treatment with low molecular weight heparin would be appropriate.

'Venous thromboembolic diseases: diagnosis, management and thrombophilia testing' Clinical guideline [CG144]. The National Institute for Health and Care Excellence, June 2012.

👍 👎 Discuss (4) Improve

Next question >

Pulmonary embolism: investigation ★

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:
- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

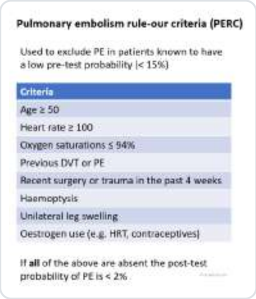
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
    - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	**Points**
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1

Clinical feature	Points
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores

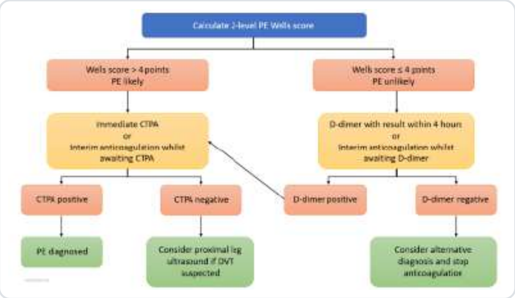
- PE likely - more than 4 points
- PE unlikely - 4 points or less

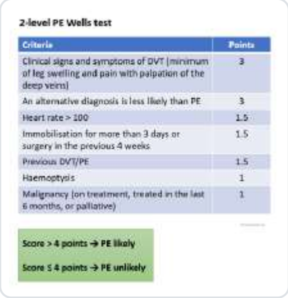
If a PE is 'likely' (more than 4 points)

- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
  - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
  - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
  - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
  - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
  - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points
PE likely
Wells score ≤ 4 points
PE unlikely
Immediate CTPA
or
Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours
or
Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

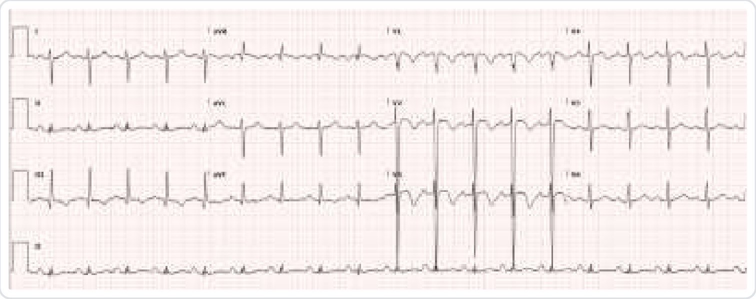
Some other points

D-dimers

- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen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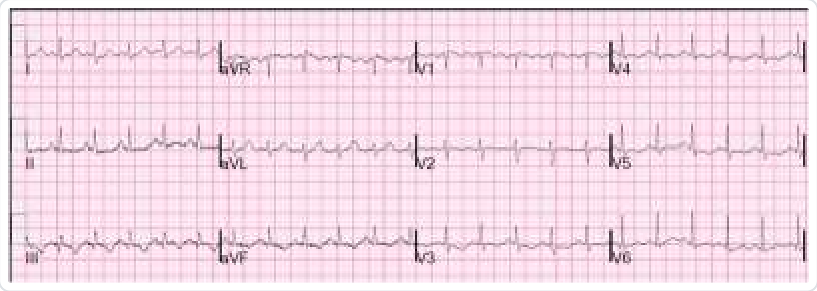
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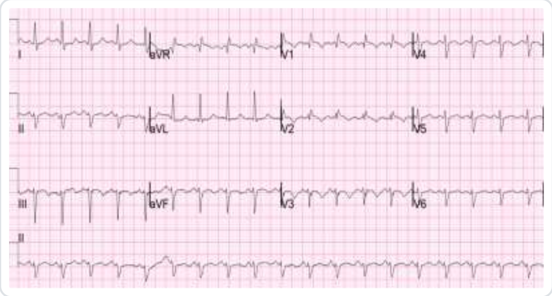
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II

Chest x-ray

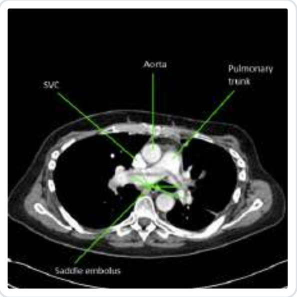
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

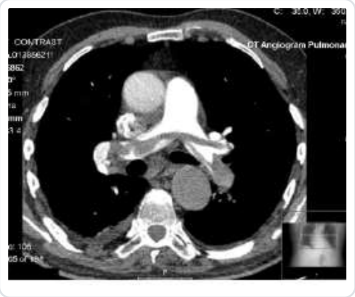
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus


CONTRAST
CT Angiogram Pulmonar

1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

MDcalc 4 0

PERC Rule for Pulmonary Embolism

NICE 👍 2 👎 1

2020 Venous thromboembolism guidelines

Life in the Fast Lane 👍 1 👎 1

The ECG in Pulmonary Embolism

MDcalc 👍 1 👎 1

Wells' Criteria for Pulmonary Embolism

Suggest link Report broken link

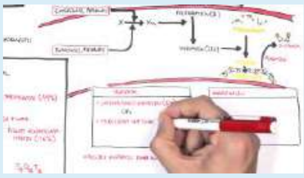
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

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Score: **21.2%**

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Question 100 of 129

A 69-year-old gentleman is reviewed in cardiology clinic. He has been suffering from angina for five years and despite optimal medical management he finds that his symptoms are worsening. He has a past medical history of type 2 diabetes mellitus, gout and hypertension. He is currently taking felodipine, atenolol, atorvastatin, aspirin, ramipril and metformin. He has recently undergone an angiogram which has delineated triple vessel disease. He is very keen to optimise his survival chances as he is concerned about dying of a heart attack.

What intervention would have the greatest survival advantage?

- ○ Aggresive control of blood pressure, cholesterol and diabetes ×
- ○ Percutaneous coronary intervention (PCI) with insertion of stents ×
- ○ Coronary arterial bypass graft (CABG) ×
- ○ No intervention will give a further survival advantage ×
- ○ Heart transplant ×

Submit answer

Reference ranges

Score: **21.2%**

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What intervention would have the greatest survival advantage?

Aggresive control of blood pressure, cholesterol and diabetes	10%
Percutaneous coronary intervention (PCI) with insertion of stents	10%
Coronary arterial bypass graft (CABG)	76%
No intervention will give a further survival advantage	2%
Heart transplant	1%

The correct answer is coronary arterial bypass graft (CABG). This is a gentleman with severe ischaemic heart disease and angina. The greatest benefits are in either PCI or CABG as they address the underlying problem; reduced coronary artery blood flow. CABG is favoured over PCI in the case of people who are over 65 years old, have diabetes or have triple vessel disease. This patient has all three and therefore is a better option, but both options should be discussed in an MDT setting and with the patient.

Source:

'Unstable angina and NSTEMI: early management' Clinical guideline [CG94]. The National Institute for Health and Care Excellence, March 2010.

Discuss (4) Improve

Next question

Angina pectoris: drug management ★

The management of stable angina comprises lifestyle changes, medication, percutaneous coronary intervention and surgery. NICE produced guidelines in 2011 covering the management of stable angina

Medication

- all patients should receive aspirin and a statin in the absence of any contraindication
- sublingual glyceryl trinitrate to abort angina attacks
- NICE recommend using either a beta-blocker or a calcium channel blocker first-line based on 'comorbidities, contraindications and the person's preference'
- if a calcium channel blocker is used as monotherapy a rate-limiting one such as verapamil or diltiazem should be used
- if used in combination with a beta-blocker then use a longer-acting dihydropyridine calcium channel blocker (e.g. amlodipine, modified-release nifedipine)
    - remember that beta-blockers should not be prescribed concurrently with verapamil (risk of complete heart block)
- if there is a poor response to initial treatment then medication should be increased to the maximum tolerated dose (e.g. for atenolol 100mg od)
- if a patient is still symptomatic after monotherapy with a beta-blocker add a calcium channel blocker and vice versa
- if a patient is on monotherapy and cannot tolerate the addition of a calcium channel blocker or a beta-blocker then consider one of the following drugs:
    - a long-acting nitrate
    - ivabradine
    - nicorandil
    - ranolazine
- if a patient is taking both a beta-blocker and a calcium-channel blocker then only add a third drug whilst a patient is awaiting assessment for PCI or CABG

Nitrate tolerance

- many patients who take nitrates develop tolerance and experience reduced efficacy
- NICE advises that patients who take standard-release isosorbide mononitrate should use an asymmetric dosing interval to maintain a daily nitrate-free time of 10-14 hours to minimise the development of nitrate tolerance
- this effect is not seen in patients who take once-daily modified-release isosorbide mononitrate

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 22 👎 24

2011 Stable angina guidelines

Clinical Knowledge Summaries 👍 22 👎 20

Angina guidelines

SIGN 👍 25 👎 22

2018 Management of stable angina

Royal College of Physicians 👍 32 👎 16

2013 Pharmacological treatment of chronic stable angina pectoris

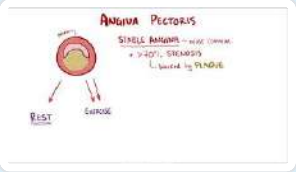
Suggest link Report broken link

Media

MIDLINE STERNOTOMY SCAR
ATHEROSCLEROSIS
ANGINA
CT CORONARY ANGIOGRAM
SAPHENOUS VEIN HARVESTING SCAR
GTN SPRAY

Stable Angina: Visual Explanation for Students

Zero To Finals - YouTube 👍 9 👎 1


ANGINA PECTORIS
STABLE ANGINA
REST
EXERCISE

Angina pectoris (stable, unstable, prinzmetal, vasospastic)

Osmosis - YouTube 👍 2 👎 1

Report broken media

Score: **21%**

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Question 101 of 129

A 64-year-old woman presents to the emergency department with breathlessness. She has been feeling progressively breathless over the last two weeks with marked reduction in her exercise tolerance. She now finds that even a few steps make her short of breath whilst normally she would be able to walk to her local shops and back. She finds that she cannot lie down as it makes her feel worse. She has a past medical history of hypertension, previous myocardial infarction and asthma.

On examination, she has a raised JVP, bilateral crepitations on auscultation, reduced air entry in both bases and dull percussion. On her chest X-ray, there is evidence of pulmonary oedema and bilateral pleural effusions. What is the most appropriate management plan?

- ○ Treat medically with diuretics ×
- ○ Pleural aspirate and if exudate insert chest drain ×
- ○ Insert chest drain on one side ×
- ○ Insert chest drain on both sides ×
- ○ Non-invasive ventilation ×

Submit answer

Reference ranges

Score: **21%**

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Question 101 of 129 ✗

A 64-year-old woman presents to the emergency department with breathlessness. She has been feeling progressively breathless over the last two weeks with marked reduction in her exercise tolerance. She now finds that even a few steps make her short of breath whilst normally she would be able to walk to her local shops and back. She finds that she cannot lie down as it makes her feel worse. She has a past medical history of hypertension, previous myocardial infarction and asthma.

On examination, she has a raised JVP, bilateral crepitations on auscultation, reduced air entry in both bases and dull percussion. On her chest X-ray, there is evidence of pulmonary oedema and bilateral pleural effusions. What is the most appropriate management plan?

Treat medically with diuretics	90%
Pleural aspirate and if exudate insert chest drain	3%
Insert chest drain on one side	1%
Insert chest drain on both sides	1%
Non-invasive ventilation	5%

The correct answer is to treat medically with diuretics. The history, examination and imaging all strongly suggest the underlying cause for her shortness of breath to be due to heart failure. The presence of the pleural effusions are therefore most likely to be transudates and it is reasonable to first treat the underlying cause before attempting to drain one or both of the effusions. If medical management with diuretics and nitrates failed to control this acute exacerbation of heart failure then non-invasive ventilation would be appropriate.

Discuss (4) Improve

Next question ›

Heart failure: acute management ★

Recommended treatments for all patients

- IV loop diuretics
    - e.g. furosemide or bumetanide

Possible additional treatments

- oxygen
    - this should be given in line with British Thoracic Society guidelines, i.e. keep oxygen saturations at 94-98%
- vasodilators
    - nitrates should not be routinely given to all patients
    - they may, however, have a role if concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease
    - the major side-effect/contraindication is hypotension

Patients with respiratory failure

- CPAP

Patients with hypotension (e.g. < 85 mmHg)/cardiogenic shock

- this can be a difficult scenario to manage
    - some of the treatments typically used for acute heart failure (e.g. loop diuretics and nitrates) may exacerbate the hypotension
- inotropic agents
    - e.g. dobutamine
    - should be considered for patients with severe left ventricular dysfunction who have potentially reversible cardiogenic shock
- vasopressor agents
    - e.g. norepinephrine

    - normally only used if insufficient response to inotropes and evidence of end-organ hypoperfusion
- mechanical circulatory assistance
    - e.g. intra-aortic balloon counterpulsation or ventricular assist devices

Other treatments

- opiates
    - NICE state 'do not routinely offer opiates to people with acute heart failure'
    - they were previously used routinely and help to reduce dyspnoea/distress in patients
    - however some studies have suggested increased morbidity in patients given opiates

General points

- regular medication for heart failure such as beta-blockers ACE-inhibitors should be continued
    - beta-blockers should only be stopped if the patient has heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock

Next question

Textbooks

High-yield textbook

Extended textbook

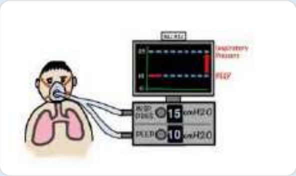
Links

NICE 👍 6 👎 9

2014 Acute heart failure: diagnosis and management

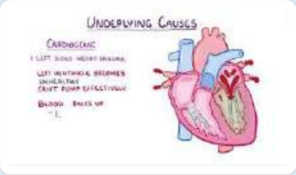
Suggest link Report broken link

Media


Inspiratory Pressure
PEEP
15 cmH2O
10 cmH2O

CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 👍 17 👎 0


Underlying Causes
Cardiogenic

Pulmonary oedema

Osmosis - YouTube 👍 4 👎 0

Report broken media

Score: **20.8%**

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Question 102 of 129

A 45-year-old gentleman is advised by his GP to attend hospital following a routine blood test demonstrating an increase in creatinine two weeks following a dose increase in his ramipril. His ramipril was increased from 5mg daily to 5mg twice a day.

He has a letter which demonstrates investigation results, shown below. He reports no new symptoms. He reports that home monitoring of blood pressure has shown his control to be better.

	21/11/2016	6/11/2016
Na^+	140 mmol/l	138 mmol/l
K^+	4.5 mmol/l	4.1 mmol/l
Urea	5.5 mmol/l	5.4 mmol/l
Creatinine	110 µmol/l	92 µmol/l

How should his ramipril be managed and monitored?

- ○ Continue ramipril and repeat U&Es in 1-2 weeks ×
- ○ Reduced the dose of ramipril and repeat U&Es in 1-2 weeks ×
- ○ Stop ramipril and repeat U&Es in 1-2 weeks ×
- ○ Stop ramipril and arrange for an urgent out-patient imaging to exclude renal artery stenosis ×
- ○ Continue ramipril and repeat U&Es within 48 hours ×

Submit answer

Reference ranges

Score: **20.8%**

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Question 102 of 129 ✗

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K^+	4.5 mmol/l	4.1 mmol/l
Urea	5.5 mmol/l	5.4 mmol/l
Creatinine	110 µmol/l	92 µmol/l

How should his ramipril be managed and monitored?

Continue ramipril and repeat U&Es in 1-2 weeks 90%

Reduced the dose of ramipril and repeat U&Es in 1-2 weeks 3%

Stop ramipril and repeat U&Es in 1-2 weeks 2%

Stop ramipril and arrange for an urgent out-patient imaging to exclude renal artery stenosis 3%

Continue ramipril and repeat U&Es within 48 hours 2%

The correct answer is to continue ramipril and repeat U&Es in 1-2 weeks. Up to an increase in creatinine of 30% can be tolerated following the start or increase in an ACE-inhibitor such as ramipril, but U&Es should be repeated in 1-2 weeks. If the increase is greater than 30% then the ACE-inhibitor should be stopped.

Source:

'Chronic Kidney Disease in Adults: Assessment and Management (CG182).' National Institute for Health and Care Excellence (2014)

👍 👎 Discuss (2) Improve

Next question >

ACE inhibitors ★

Angiotensin-converting enzyme (ACE) inhibitors are now the established first-line treatment in younger patients with hypertension and are also extensively used to treat heart failure. They are known to be less effective in treating hypertensive Afro-Caribbean patients. ACE inhibitors are also used to treat diabetic nephropathy and have a role in the secondary prevention of ischaemic heart disease.

Mechanism of action:

- inhibits the conversion angiotensin I to angiotensin II
    - → decrease in angiotensin II levels → to vasodilation and reduced blood pressure
    - → decrease in angiotensin II levels → reduced stimulation for aldosterone release → decrease in sodium and water retention by the kidneys
- renoprotective mechanism
    - angiotensin II constricts the efferent glomerular arterioles
    - ACE inhibitors therefore lead to dilation of the efferent arterioles → reduced glomerular capillary pressure → decreased mechanical stress on the delicate filtration barriers of the glomeruli
    - this is particularly important in diabetic nephropathy
- ACE inhibitors are activated by phase 1 metabolism in the liver

Side-effects:

- cough
    - occurs in around 15% of patients and may occur up to a year after starting treatment
    - thought to be due to increased bradykinin levels
- angioedema: may occur up to a year after starting treatment
- hyperkalaemia

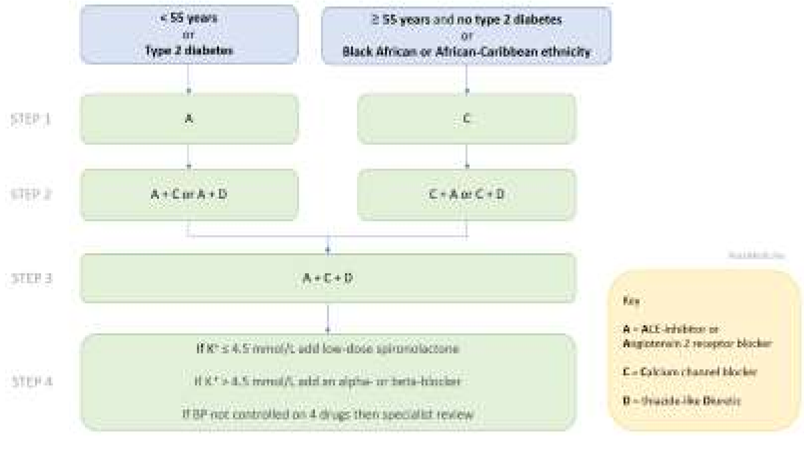
- first-dose hypotension: more common in patients taking diuretics

Cautions and contraindications
- pregnancy and breastfeeding - avoid
- renovascular disease - may result in renal impairment
- aortic stenosis - may result in hypotension
- hereditary of idiopathic angioedema
- specialist advice should be sought before starting ACE inhibitors in patients with a potassium >= 5.0 mmol/L

Interactions
- patients receiving high-dose diuretic therapy (more than 80 mg of furosemide a day)
    - significantly increases the risk of hypotension

Monitoring
- urea and electrolytes should be checked before treatment is initiated and after increasing the dose
    - a rise in the creatinine and potassium may be expected after starting ACE inhibitors
    - acceptable changes are an increase in serum creatinine, up to 30% from baseline and an increase in potassium up to 5.5 mmol/l.
    - significant renal impairment may occur in patients who have undiagnosed bilateral renal artery stenosis

Examples of common ACE inhibitors
- ramipril
- enalapril
- lisinopril


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K+ ≤ 4.5 mmol/L add low-dose spironolactone
If K+ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2014 Chronic kidney disease guidelines	👍 53 👎 34
BNF ACE inhibitors	👍 50 👎 25

Suggest link

Report broken link

Score: **20.6%**

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101	✗
102	✗

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Question 103 of 129

A 75-year-old gentleman with a background of type II diabetes mellitus, ischaemic heart disease (IHD) and New York Heart Association (NYHA) III heart failure presents to your clinic with continuing fatigue and shortness of breath on minimal exertion. He currently takes Aspirin 75mg, Bisoprolol 10mg, Ramipril 5mg, Furosemide 40mg twice daily and Spironolactone 25mg. His electrocardiograph demonstrates left bundle branch block (old) and his ejection fraction is 32% measured by echocardiography at the clinic today. His chest x-ray shows an enlarged cardio-thoracic ratio but no obvious pulmonary oedema. Blood tests taken with his general practitioner this week are as follows:

Haemoglobin	110g/l
White cell count	11.0 *10^9
Neutrophils	7.2 *10^9
Platelets	240 *10^9
Na+	137 mmol/l
K+	4.2 mmol/l
Urea	9 mmol/l
Creatinine	125 μmol/l

What evidence-based treatment, if any, is available to treat this gentleman's cardiac failure?

- ○ Increase dose of Spironolactone to 50mg once daily
- ○ Increase dose of Furosemide to 80mg twice daily
- ○ Cardiac-resynchronisation therapy
- ○ Angiography +/- coronary stenting
- ○ He is already on maximal treatment

Submit answer

A 75-year-old gentleman with a background of type II diabetes mellitus, ischaemic heart disease (IHD) and New York Heart Association (NYHA) III heart failure presents to your clinic with continuing fatigue and shortness of breath on minimal exertion. He currently takes Aspirin 75mg, Bisoprolol 10mg, Ramipril 5mg, Furosemide 40mg twice daily and Spironolactone 25mg. His electrocardiograph demonstrates left bundle branch block (old) and his ejection fraction is 32% measured by echocardiography at the clinic today. His chest x-ray shows an enlarged cardio-thoracic ratio but no obvious pulmonary oedema. Blood tests taken with his general practitioner this week are as follows:

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Platelets	240 *10^9
Na+	137 mmol/l
K+	4.2 mmol/l
Urea	9 mmol/l
Creatinine	125 µmol/l

What evidence-based treatment, if any, is available to treat this gentleman's cardiac failure?

Increase dose of Spironolactone to 50mg once daily	17%
Increase dose of Furosemide to 80mg twice daily	3%
Cardiac-resynchronisation therapy	75%
Angiography +/- coronary stenting	2%
He is already on maximal treatment	2%

NICE guidance has recommended that cardiac resynchronisation therapy can be offered to patients who fulfil all of the following criteria:

- NYHA III or IV heart failure
- Ejection fraction of <35%
- The heart is beating regularly with evidence of electrical conduction disease
- They are medication that is most effective for them

This patient is on appropriate doses of secondary-prevention and heart failure medications and there is no evidence of an acute coronary syndrome but the previous IHD is likely the cause of his cardiac failure. He satisfies all of the above criteria for cardiac resynchronisation and this has a good evidence-base.

Discuss (22) Improve

Next question >

Chronic heart failure: non-drug management ★

Cardiac resynchronisation therapy

- for patients with heart failure and wide QRS
- biventricular pacing
- improved symptoms and reduced hospitalisation in NYHA class III patients

Exercise training

- improves symptoms but not hospitalisation/mortality

AI + Q 123

Next question >

B *I* A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 0 👎 0

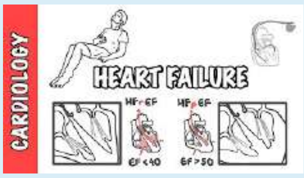
2018 Chronic heart failure guidelines

NICE 👍 10 👎 1

Cardiac resynchronisation therapy for the treatment of heart failure

Suggest link Report broken link

Media


CARDIOLOGY
HEART FAILURE
HFrEF
HFpEF
EF<40
EF>50

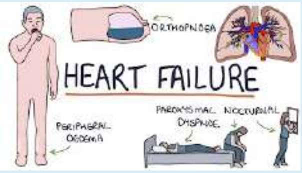
Heart failure with reduced and preserved ejection fraction

Armando Hasudungan - YouTube 👍 0 👎 0

Osmosis.org
Heart Failure
Medical Student Review
WATCH

Heart failure

Osmosis - YouTube 👍 4 👎 1


ORTHOPNOEA
HEART FAILURE
PAROXYSMAL NOCTURNAL DYSPNOEA
PERIPHERAL OEDEMA

Chronic Heart Failure: Visual Explanation for Students

Zero To Finals - YouTube

👍 1 👎 2

Report broken media

Score: **20.4%**

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102 ✗

103 ✗

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A 76-year-old woman is brought into the Emergency Department following an episode of loss of consciousness. On more detailed review, she reveals that she has been suffering increasingly with shortness of breath on exertion, with great difficulty in managing her shopping and cleaning.

Her past medical history include right hip fracture 6 years ago after a fall walking down the stairs; this required a total hip replacement.

She takes no regular medication and has no drug allergies.

On examination, her pulse was 80 beats per minute, blood pressure 104/89 mmHg, and respiratory rate 16 breaths per minute. She had a slow rising pulse, and on auscultation of the chest there was an ejection systolic murmur heard loudest at the aortic area, and radiating to the carotid arteries.

Electrocardiogram (ECG) shows sinus rhythm and criteria for left ventricular hypertrophy.

Chest x-ray shows clear lung fields.

Transthoracic echocardiogram:

Aortic valve area	0.8 cm^2 (3-4)
Transvalvular gradient	55 mmHg (0)

The diagnosis of aortic stenosis is explained to the patient, and she opts for open surgical valve replacement.

What other investigation is warranted before proceeding to aortic valve replacement?

○ Exercise tolerance testing ×

○ Dopamine stress echocardiogram ×

○ 24 hour holter ECG ×

○ Transoesophageal echocardiogram ×

○ Coronary angiography ×

Submit answer

Reference ranges ⌄

Score: **20.4%**

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Question 104 of 129 ✓

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Transvalvular gradient	55 mmHg (0)

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What other investigation is warranted before proceeding to aortic valve replacement?

Exercise tolerance testing 4%

Dopamine stress echocardiogram 4%

24 hour holter ECG	3%
Transoesophageal echocardiogram	9%
Coronary angiography	81%

The valve area <1 cm^2 and transvalvular gradient >50 mmHg on transthoracic echocardiogram confirm the diagnosis of severe aortic stenosis, and the patient has opted for open surgical valve replacement.

Patients undergoing open surgical valve replacement should first undergo coronary angiography to exclude any coronary stenosis that could simultaneously be treated with bypass grafting.

This patient has undergone an 12 lead ECG and transthoracic echocardiogram. Further ECG or echocardiogram studies are not usually necessary.

Discuss (5) Improve

Next question >

Aortic stenosis

Clinical features of symptomatic disease
- chest pain
- dyspnoea
- syncope / presyncope (e.g. exertional dizziness)
- murmur
  - an **ejection systolic murmur** (ESM) is classically seen in aortic stenosis
  - classically radiates to the carotids
  - this is decreased following the Valsalva manoeuvre

Features of severe aortic stenosis
- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2

- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in older patients > 65 years)
- bicuspid aortic valve (most common cause in younger patients < 65 years)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is a general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 40 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- options for aortic valve replacement (AVR) include:
  - surgical AVR is the treatment of choice for young, low/medium operative risk patients. Cardiovascular disease may coexist. For this reason, an angiogram is often done prior to surgery so that the procedures can be combined
  - transcatheter AVR (TAVR) is used for patients with a high operative risk
- balloon valvuloplasty
  - may be used in children with no aortic valve calcification
  - in adults limited to patients with critical aortic stenosis who are not fit for valve replacement

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 12 👎 17

2021 Valvular heart disease guidelines

American Heart Association 👍 22 👎 10

Guidelines on the management of valvular heart disease

Suggest link

Report broken link

Media

Aortic stenosis murmur - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 5 👎 1

AORTIC VALVE DISEASE
OSMOSIS.org

Aortic valve disease (bicuspid, tricuspid) - stenosis, regurgitation

Osmosis - YouTube 👍 5 👎 2

AORTIC STENOSIS

Aortic stenosis

Armando Hasudungan - YouTube 👍 5 👎 2

Report broken media

Score: **21.2%**

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104 ✓

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Question 105 of 129

A 50-year-old man is recovering 12 hours after a stent placement for a ST elevation myocardial infarction in which there was a small stenosis of the circumflex artery. He has returned to the coronary care unit and you are called to see him due to changes on the cardiac monitor.

There are runs of broad complexes lasting 3-10 seconds at a rate of 80 per minute. He is asymptomatic and on waking is able to tell you that he has no chest pain or palpitations.

Na^+	135 mmol/l
K^+	3.2 mmol/l
Urea	5.3 mmol/l
Creatinine	89 µmol/l
Magnesium	0.61mmol/l (normal range 0.6-1.0)
Troponin	3000pg/ml (normal range <20)
ECG	Broad complexes at 73 beats per minute running in sets of 3-15 with normal sinus rhythm between. PR 150ms, QTc 450ms. No ST elevation at present

What action should be taken?

- ○ Reassure
- ○ IV magnesium sulfate
- ○ Transcutaneous pacing
- ○ DC cardioversion
- ○ Amiodarone

Submit answer

Reference ranges ⌵

Score: **21.2%**

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103 ✗

104 ✓

105 -

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A 50-year-old man is recovering 12 hours after a stent placement for a ST elevation myocardial infarction in which there was a small stenosis of the circumflex artery. He has returned to the coronary care unit and you are called to see him due to changes on the cardiac monitor.

There are runs of broad complexes lasting 3-10 seconds at a rate of 80 per minute. He is asymptomatic and on waking is able to tell you that he has no chest pain or palpitations.

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Urea	5.3 mmol/l
Creatinine	89 µmol/l
Magnesium	0.61mmol/l (normal range 0.6-1.0)
Troponin	3000pg/ml (normal range <20)
ECG	Broad complexes at 73 beats per minute running in sets of 3-15 with normal sinus rhythm between. PR 150ms, QTc 450ms. No ST elevation at present

What action should be taken?

Reassure	59%
IV magnesium sulfate	30%
Transcutaneous pacing	2%
DC cardioversion	1%
Amiodarone	7%

Accelerated idioventricular rhythm is common and unconcerning following recent MI

Important for me Less important

This gentleman has an MI that has been successfully treated and is recovering well. He is at risk of malignant arrhythmias like ventricular tachycardia during this time. However these broad complexes are not tachycardic. This phenomenon is accelerated idioventricular rhythm which commonly follows an MI in which ventricular ectopic tissue take over the pacemaker role. This usually resolves with time but can be helped by atropine if the patient is symptomatic. In this case, there are no symptoms and so the best action is to reassure the patient and continue monitoring. All other options would not be appropriate given the benign nature of this rhythm.

👍 | 👎 | 💬 Discuss (8) | Improve

Next question >

Accelerated idioventricular rhythm ★

Accelerated idioventricular rhythm (AIVR) is a benign ectopic rhythm of ventricular origin. It usually occurs following the reperfusion of an ischaemic myocardium. It has a rate of 50-110 beats per minute, which helps differentiate it from ventricular bradycardia or ventricular tachycardia.

Pathophysiology:

- Reperfusion of ischaemic tissue, electrolyte abnormalities or drug toxicity leads to an increased depolarisation rate of ventricular myocytes.
- When this depolarisation rate is faster than the rate produced by the sinoatrial node, it becomes the overriding rhythm. Therefore, sinus bradycardia by vagal excess or reduced sympathetic activity can precipitate AIVR.

AIVR has a rate of between 50 -110 beats per minute and, therefore, the patient tends to be haemodynamically stable. However, if the rate is fast or there is extensive damage to the myocardium, the patient can become unstable.

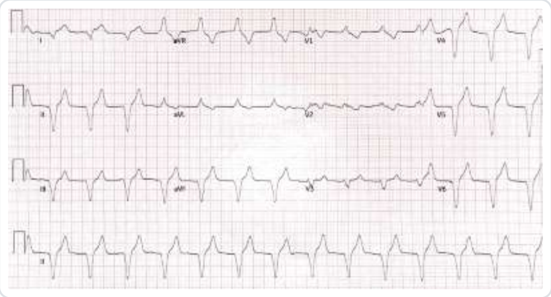
Causes:

- Reperfusion following myocardial infarction - by far the most common cause

- Beta-sympathomimetics (e.g. adrenaline)
- Drug toxicity (digoxin/cocaine)
- Electrolyte imbalance
- Cardiomyopathy, congenital heart disease, myocarditis

AIVR is diagnosed based on electrocardiography (ECG) findings:

- AIVR has a gradual onset and termination as the ventricular rate is only slightly faster than sinus rhythm. This may result in ventricular fusion beats on ECG.
- Atrioventricular dissociation - P waves present but not associated with QRS complexes.
- The ECG will show wide QRS complexes >120ms.
- Rate between 50-110 beats per minute.



Treatment:

- AIVR is usually self-limiting and therefore treatment is not necessary, however, occasionally atropine can be used to increase the sinus rate to overcome AIVR.

+ Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **21%**

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103	✗
104	✓
105	✗

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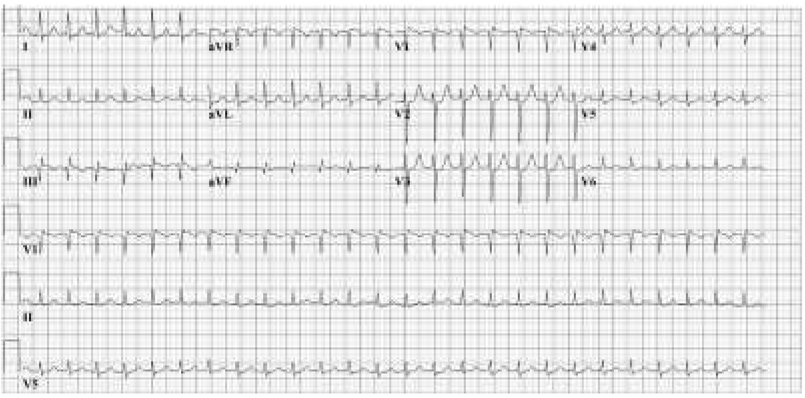
A 41-year-old woman attends the emergency department with a 6-hour history of palpitations and breathlessness. She denies chest pain or recent illness. Her past medical history includes type 1 diabetes and she smokes 15 cigarettes/day.

On examination, she has a heart rate of 140bpm with a blood pressure of 122/77mmHg. Peripheral pulses are present bilaterally with a capillary refill of < 2 seconds. On auscultation, her heart sounds are normal with vesicular breath sounds throughout.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

- ○ Atrial flutter
- ○ Sinus tachycardia
- ○ Supraventricular tachycardia
- ○ Ventricular tachycardia
- ○ Wolff-Parkinson White syndrome

Submit answer

Question 106 of 129 ✗

A 41-year-old woman attends the emergency department with a 6-hour history of palpitations and breathlessness. She denies chest pain or recent illness. Her past medical history includes type 1 diabetes and she smokes 15 cigarettes/day.

On examination, she has a heart rate of 140bpm with a blood pressure of 122/77mmHg. Peripheral pulses are present bilaterally with a capillary refill of < 2 seconds. On auscultation, her heart sounds are normal with vesicular breath sounds throughout.

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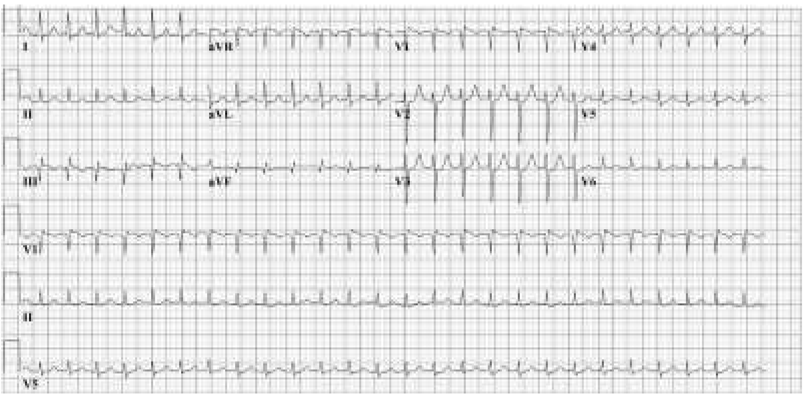


© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Atrial flutter	14%
Sinus tachycardia	12%
Supraventricular tachycardia	61%
Ventricular tachycardia	1%
Wolff-Parkinson White syndrome	12%

This patient's ECG shows a regular, narrow-complex tachycardia in keeping with supraventricular tachycardia (SVT). Given the absence of clear P waves, it is likely that the cause of this SVT is an atrioventricular nodal re-entry tachycardia (AVNRT) secondary to a re-entry circuit causing premature ventricular contraction. SVT is more common in women with a heart rate varying from between 140-280bpm. Symptoms include palpitations and breathlessness, as seen here, and may occur spontaneously or following provocation with caffeine, drugs or alcohol.

Atrial flutter is a form of supraventricular tachycardia caused by a re-entry circuit into the right atrium. This typically causes an atrial rate of 300bpm. The ventricular rate is determined by the degree of the atrioventricular block at the atrioventricular node e.g. a 2:1 block would result in a heart rate of 150bpm, whilst a 3:1 block would give a rate of 100bpm. Symptoms include palpitations or shortness of breath, whilst common ECG findings include a narrow complex tachycardia with inverted flutter waves in leads II, III and aVF, giving rise to a saw-tooth appearance on the baseline of the ECG. These are not seen here, making it a less likely diagnosis.

Sinus tachycardia is a common ECG finding in patients presenting with palpitations. However, the absence of discernable P waves makes sinus tachycardia less likely.

Ventricular tachycardia presents as a broad-complex tachycardia with widened QRS complexes. This ECG shows a narrow-complex tachycardia, making ventricular tachycardia unlikely.

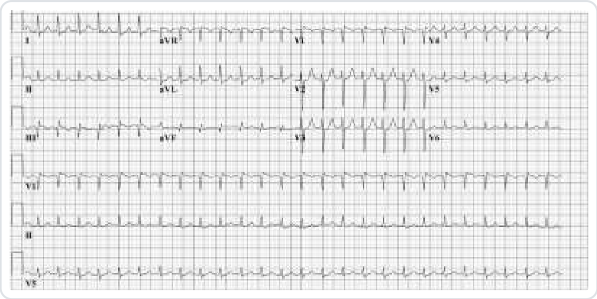
Wolff-Parkinson White (WPW) syndrome causes a tachyarrhythmia secondary to an accessory excitatory pathway stimulating premature ventricular contraction. WPW syndrome may present as palpitations, as seen in this patient. However, the ECG findings in Wolff-Parkinson White include a shortened PR interval and a slurred upstroke of the QRS complex (delta wave) causing QRS interval prolongation which is not seen here.

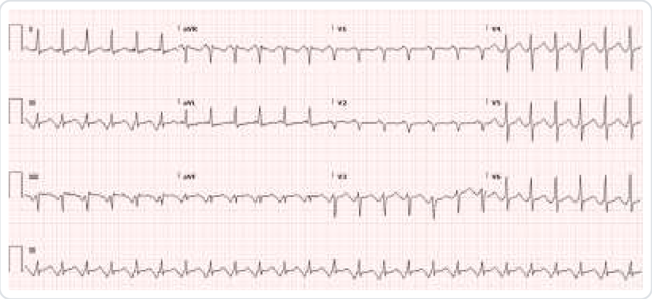
👍 | 👎 | 💬 Discuss (5) | Improve

Next question >

Supraventricular tachycardia ★

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
  - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
  - carotid sinus massage
- intravenous adenosine
  - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
  - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

2021 Tachycardia algorithm

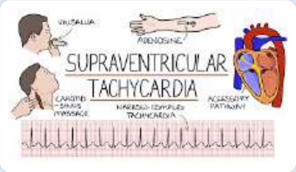
Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

Suggest link

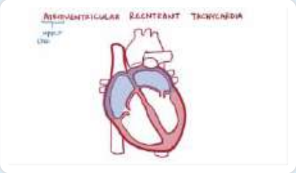
Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

Understanding Supraventricular Tachycardia (SVT)

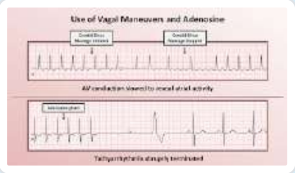
Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

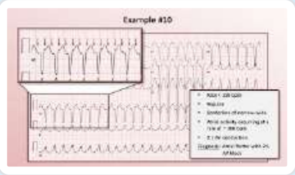
Osmosis - YouTube

👍 8 👎 0


Use of Vagal Maneuvers and Adenosine
AV conduction slowed to reveal atrial activity
Tachyarrhythmia abruptly terminated

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0



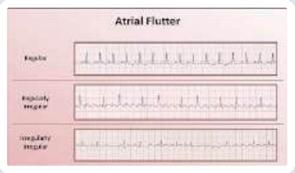
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0

ATRIOVENTRICULAR NODAL RE-ENTRANT TACHYCARDIA (AVNRT)
AVRT & AVNRT
Khan Academy

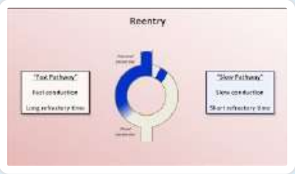
Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0


Atrial Flutter
Regular
Regularly irregular
Irregularly irregular

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **20.8%**

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101	✗
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103 ✗
104 ✓
105 ✗
106 ✗

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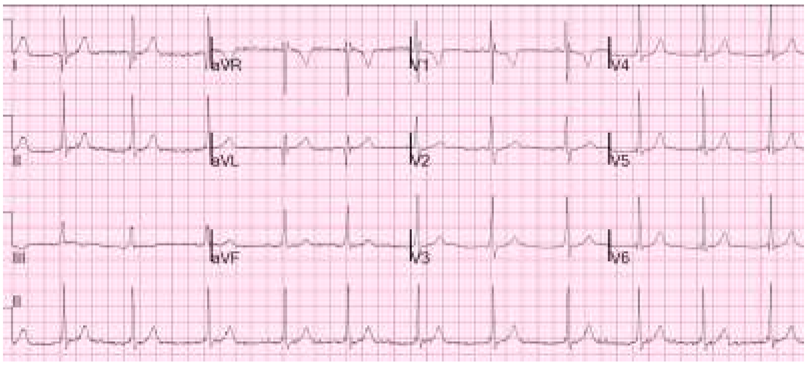
Question 107 of 129

A 40-year-old woman presents to the emergency department with a 10-day history of intermittent palpitations. She reports that her symptoms have worsened over the last 8 hours and her palpitations are now persistent and are associated with lightheadedness. She has no past medical history of note and takes no regular medications.

On examination, she appears clammy and warm to the touch. Her heart rate is 72 beats/min with a blood pressure of 125/83mmHg.

An ECG is taken (as shown below):



© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

- ○ Accelerated junctional rhythm
- ○ Atrial flutter
- ○ Normal sinus rhythm
- ○ Supraventricular tachycardia
- ○ Wolff-Parkinson white syndrome

Submit answer

Reference ranges

Score: **20.8%**

1	✗
2	✓
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101	✗
102	✗

103	✗
104	✓
105	✗
106	✗
107	-

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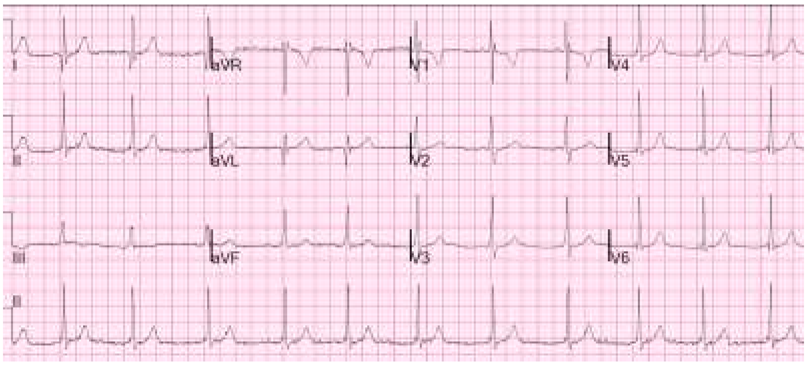


© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Accelerated junctional rhythm	14%
Atrial flutter	5%
Normal sinus rhythm	5%
Supraventricular tachycardia	3%
Wolff-Parkinson white syndrome	73%

This patient has **Wolff-Parkinson White syndrome** a pre-excitation syndrome that causes clinical features of palpitations, shortness of breath, chest pain and in severe cases collapse and shock, including cardiac arrest. Wolff-Parkinson White is caused by an accessory pathway conducting impulses from the sinoatrial node to the ventricles giving rise to an atrioventricular re-entry tachycardia. ECG features include a shortened PR interval, left axis deviation or right axis deviation (depending on the location of the accessory pathway) and a widened QRS complex from the 'delta' wave (a slurred upstroke of the QRS complex). The delta wave can be seen in this patient's ECG, along with a shortened PR interval.

Accelerated junctional rhythm is incorrect. Accelerated junctional rhythm is when the atrioventricular node conducts at a faster rate than the sinoatrial node. This gives rise to an ECG with appearances of a narrow complex rhythm, with a ventricular rate between 60-100 beats/min. Retrograde P waves can be seen. Causes include digoxin toxicity, beta-blockers and myocardial ischaemia.

Atrial flutter is incorrect. Atrial flutter is an irregular supraventricular arrhythmia. Although it can cause similar symptoms to those experienced by the patient, a characteristic ECG finding is a saw-tooth appearance of the baseline of the ECG which is not seen here.

Normal sinus rhythm is incorrect. Although the patient's rhythm is sinus with QRS complexes following a P wave. There are clear delta waves preceding the QRS complexes that are consistent with Wolff-Parkinson White syndrome.

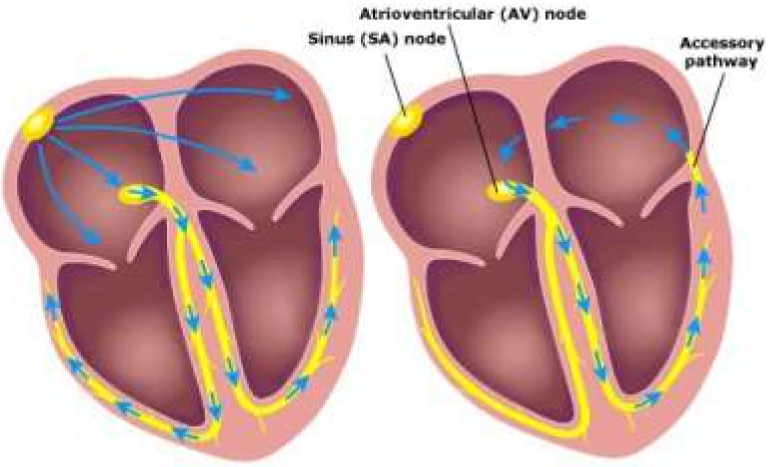
Supraventricular tachycardia (SVT) is incorrect. SVT is caused by a re-entry circuit within the atrioventricular node. This is in contrast to atrioventricular re-entry tachycardia which is caused by an accessory conducting pathway (e.g. Wolff-Parkinson White syndrome). Symptoms are similar to this patient's presentation with palpitations, shortness of breath and chest pain that may be exacerbated by caffeine or alcohol. However, the presence of a delta wave is more suggestive of an accessory conducting pathway, making supraventricular tachycardia.

👍 👎 Discuss (7) Improve

Next question >

Wolff-Parkinson White ★

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF.


Atrioventricular (AV) node
Sinus (SA) node
Accessory pathway

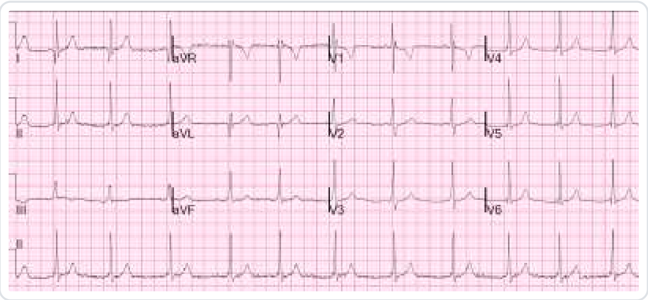
Normal electrical pathways

Abnormal electrical pathway in Wolff-Parkinson-White syndrome

Image sourced from Wikipedia

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway
  - in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation
- right axis deviation if left-sided accessory pathway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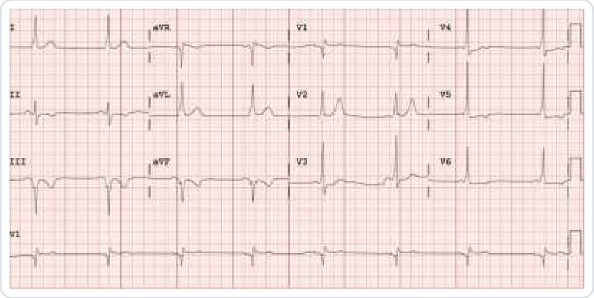
II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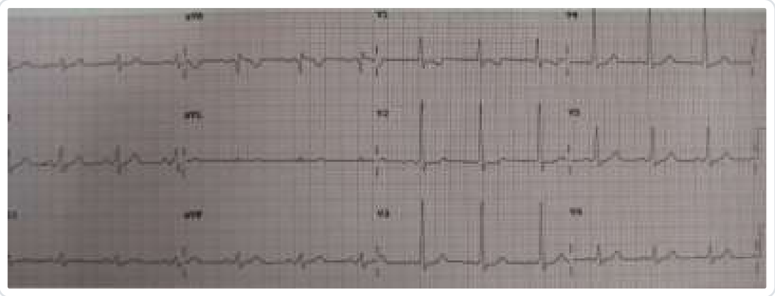

Differentiating between type A and type B

- type A (left-sided pathway): dominant R wave in V1
- type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

- HOCM
- mitral valve prolapse
- Ebstein's anomaly
- thyrotoxicosis
- secundum ASD

Management

- definitive treatment: radiofrequency ablation of the accessory pathway
- medical therapy: sotalol***, amiodarone, flecainide
  - sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Wolff-Parkinson-White syndrome

Osmosis - YouTube 👍 12 👎 2

Report broken media

Score: **21.5%**

1	✗
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107	✓

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You are asked to see a 64-year-old woman in the emergency department who has presented with a 3-day history of pleuritic chest pain and small-volume haemoptysis.

She has a past medical history of hypertension, stage 4 chronic kidney disease, previous deep venous thrombosis (DVT), and a newly diagnosed primary breast cancer awaiting multidisciplinary team discussion.

You note crackles at the right lung base. There are no added heart sounds and no limb swelling is noted.

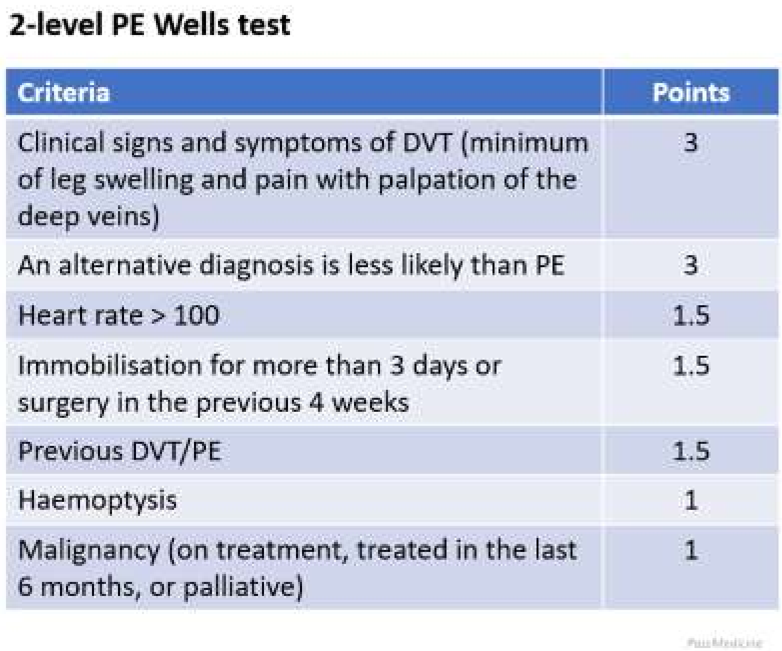
Her observations are as follows:

- Oxygen saturation: 98% on room air
- Respiratory rate: 18 per minute
- Heart rate: 104bpm
- Blood pressure 110/57 mmHg

Her blood results return as below.

Hb	116 g/L	Male: (135-180) Female: (115 - 160)
Platelets	181 * 10^9/L	(150 - 400)
WBC	6 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	6.1 mmol/L	(3.5 - 5.0)
Urea	13.6 mmol/L	(2.0 - 7.0)
Creatinine	382 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)

Chest x-ray shows no obvious abnormality.



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Of the options given, which is the most appropriate next step?

- ○ Arrange for CT pulmonary angiogram
- ○ Arrange for Doppler ultrasound
- ○ Arrange for V/Q Scan
- ○ Arrange for d-dimer
- ○ Arrange for echocardiography

Submit answer

Reference ranges

Score: **21.5%**

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You are asked to see a 64-year-old woman in the emergency department who has presented with a 3-day history of pleuritic chest pain and small-volume haemoptysis.

She has a past medical history of hypertension, stage 4 chronic kidney disease, previous deep venous thrombosis (DVT), and a newly diagnosed primary breast cancer awaiting multidisciplinary team discussion.

You note crackles at the right lung base. There are no added heart sounds and no limb swelling is noted.

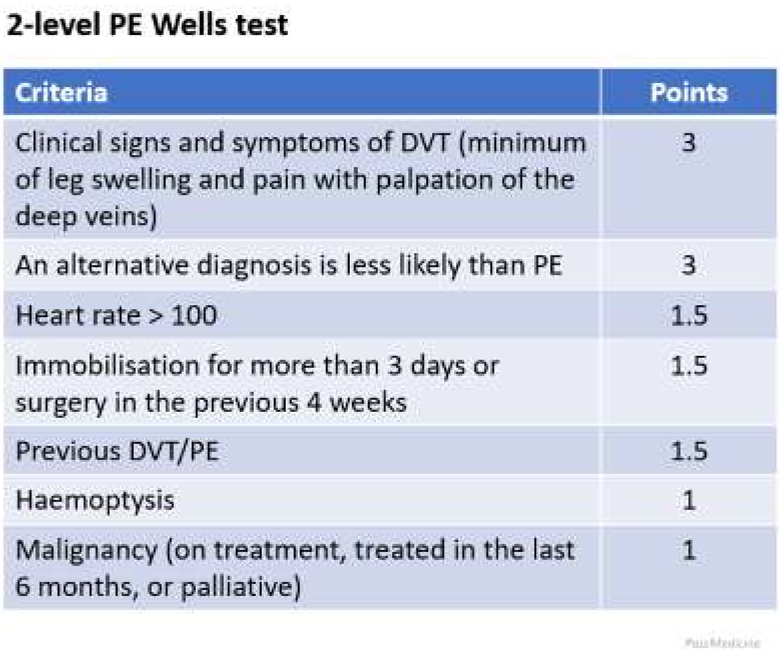
Her observations are as follows:

- Oxygen saturation: 98% on room air
- Respiratory rate: 18 per minute
- Heart rate: 104bpm
- Blood pressure 110/57 mmHg

Her blood results return as below.

Hb	116 g/L	Male: (135-180) Female: (115 - 160)
Platelets	181 * 10^9/L	(150 - 400)
WBC	6 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	6.1 mmol/L	(3.5 - 5.0)
Urea	13.6 mmol/L	(2.0 - 7.0)
Creatinine	382 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)

Chest x-ray shows no obvious abnormality.



2-level PE Wells test

Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Of the options given, which is the most appropriate next step?

Arrange for CT pulmonary angiogram	57%
Arrange for Doppler ultrasound	7%
Arrange for V/Q Scan	29%
Arrange for d-dimer	5%
Arrange for echocardiography	2%

This patient has a Wells score of 8, given that it is the most likely diagnosis, she has active malignancy, a history of previous DVT, a heart rate >100, and is presenting with haemoptysis. As they have scored more than four, pulmonary embolism (PE) would be considered likely in this patient, and therefore merits further imaging to further evaluate this, without the need for a d-dimer.

A **V/Q Scan** would be the investigation of choice for confirming a diagnosis of PE given this patient's renal function.

A **CT pulmonary angiogram** would have been the recommended initial lung-imaging modality for non-massive PE if not for being contraindicated by the patient's renal function.

Echocardiogram can be used in patients who are haemodynamically unstable, but as this is not the case here, it is not the initial investigation of choice.

Arranging for Doppler ultrasound is incorrect given there are no symptoms suggesting a current DVT.

D-dimer would be the appropriate next step for risk stratification if the patient had a Wells score of less than four.

👍 👎 Discuss (4) Improve

Next question >

Pulmonary embolism: investigation

We know from experience that few patients (around 10%) present with the textbook triad of pleuritic chest pain, dyspnoea and haemoptysis. Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So which features make pulmonary embolism more likely?

The PIOPED study[1] in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.

The relative frequency of common clinical signs is shown below:
- Tachypnea (respiratory rate >20/min) - 96%
- Crackles - 58%
- Tachycardia (heart rate >100/min) - 44%
- Fever (temperature >37.8°C) - 43%

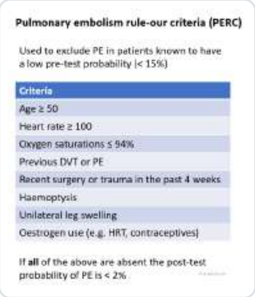
It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea.

All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a chest x-ray to exclude other pathology.

Pulmonary embolism rule-out criteria (PERC)

NICE updated their guidelines on the investigation and management of venous thromboembolism (VTE) in 2020. One of the key changes was the use of the pulmonary embolism rule-out criteria (the **PERC** rule)

- a copy of criteria can be found in the image below
- **all** the criteria must be absent to have negative PERC result, i.e. rule-out PE
- this should be done when you think there is a low pre-test probability of PE, but want more reassurance that it isn't the diagnosis
  - this low probability is defined as < 15%, although it is clearly difficult to quantify such judgements
- a negative PERC reduces the probability of PE to < 2%
- if your suspicion of PE is greater than this then you should move straight to the 2-level PE Wells score, without doing a PERC



Pulmonary embolism rule-our criteria (PERC)

Used to exclude PE in patients known to have a low pre-test probability (< 15%)

Criteria
Age ≥ 50
Heart rate ≥ 100
Oxygen saturations ≤ 94%
Previous DVT or PE
Recent surgery or trauma in the past 4 weeks
Haemoptysis
Unilateral leg swelling
Oestrogen use (e.g. HRT, contraceptives)

If **all** of the above are absent the post-test probability of PE is < 2%

2-level PE Wells score

If a PE is suspected a 2-level PE Wells score should be performed:

Clinical feature	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100 beats per minute	1.5

Clinical feature	**Points**
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Clinical probability simplified scores

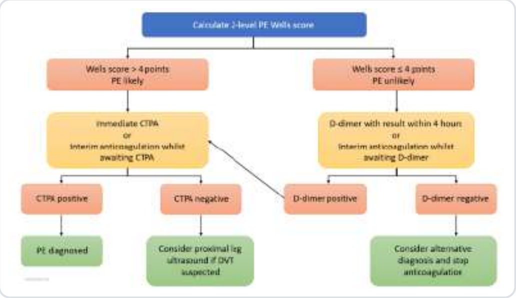
- PE likely - more than 4 points
- PE unlikely - 4 points or less

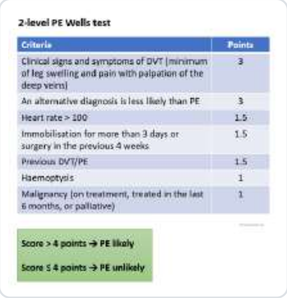
If a PE is 'likely' (more than 4 points)

- arrange an immediate computed tomography pulmonary angiogram (CTPA)
- If there is a delay in getting the CTPA then interim therapeutic anticoagulation should be given until the scan is performed.
    - interim therapeutic anticoagulation used to mean giving low-molecular-weight heparin
    - NICE updated their guidance in 2020. They now recommend using an anticoagulant that can be continued if the result is positive.
    - this means normally a direct oral anticoagulant (DOAC) such as apixaban or rivaroxaban
- if the CTPA is positive then a PE is diagnosed
- if the CTPA is negative then consider a proximal leg vein ultrasound scan if DVT is suspected

If a PE is 'unlikely' (4 points or less)

- arranged a D-dimer test
    - if positive arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give interim therapeutic anticoagulation until the scan is performed
    - if negative then PE is unlikely - stop anticoagulation and consider an alternative diagnosis


Calculate 2-level PE Wells score
Wells score > 4 points
PE likely
Wells score ≤ 4 points
PE unlikely
Immediate CTPA
or
Interim anticoagulation whilst awaiting CTPA
D-dimer with result within 4 hours
or
Interim anticoagulation whilst awaiting D-dimer
CTPA positive
CTPA negative
D-dimer positive
D-dimer negative
PE diagnosed
Consider proximal leg ultrasound if DVT suspected
Consider alternative diagnosis and stop anticoagulation



2-level PE Wells test

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Score > 4 points → PE likely

Score ≤ 4 points → PE unlikely

CTPA or V/Q scan?

The consensus view from the British Thoracic Society and NICE guidelines is as follows:

- CTPA is now the recommended initial lung-imaging modality for non-massive PE. Advantages compared to V/Q scans include speed, easier to perform out-of-hours, a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
- if the CTPA is negative then patients do not need further investigations or treatment for PE
- V/Q scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease. V/Q scanning is also the investigation of choice if there is renal impairment (doesn't require the use of contrast unlike CTPA)

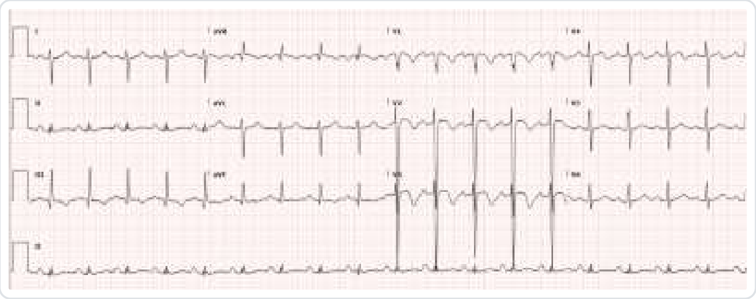
Some other points

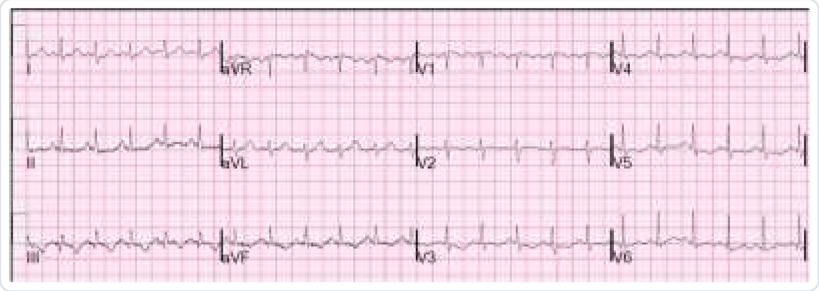
D-dimers

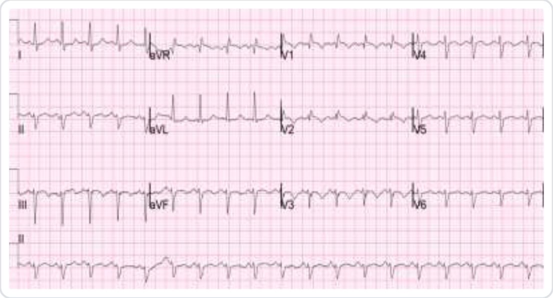
- sensitivity = 95-98%, but poor specificity
- age-adjusted D-dimer levels should be considered for patients > 50 years

ECG

- the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - 'S1Q3T3'. However, this change is seen in no more than 20% of patients
- right bundle branch block and right axis deviation are also associated with PE
- sinus tachycardia may also be seen







Chest x-ray

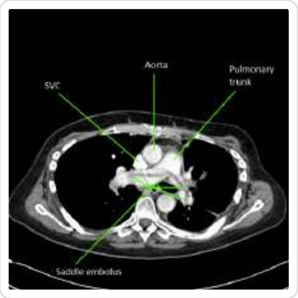
- a chest x-ray is recommended for all patients to exclude other pathology
- however, it is typically normal in PE
- possible findings include a wedge-shaped opacification

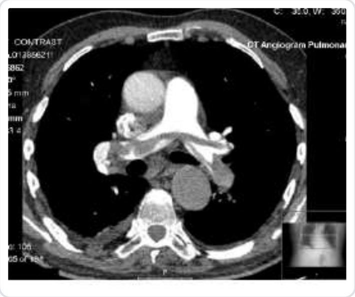
V/Q scan

- sensitivity of around 75% and specificity of 97%
- other causes of mismatch in V/Q include old pulmonary embolisms, AV malformations, vasculitis, previous radiotherapy
- COPD gives matched defects

CTPA

- peripheral emboli affecting subsegmental arteries may be missed


Aorta
Pulmonary trunk
SVC
Saddle embolus



1. Clinical Characteristics of Patients with Acute Pulmonary Embolism(Data from PIOPED II) Am J Med. Oct 2007; 120(10): 871879.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

MDcalc 👍 4 👎 0

PERC Rule for Pulmonary Embolism

NICE 👍 2 👎 1

2020 Venous thromboembolism guidelines

Life in the Fast Lane 👍 1 👎 1

The ECG in Pulmonary Embolism

MDcalc 👍 1 👎 1

Wells' Criteria for Pulmonary Embolism

Suggest link Report broken link

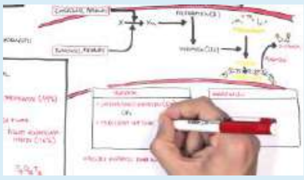
Media

Pulmonary embolism

Osmosis - YouTube 👍 2 👎 0

Pulmonary Embolism PART I

Armando Hasudungan - YouTube 👍 5 👎 1



Pulmonary Embolism PART II

Armando Hasudungan - YouTube 👍 4 👎 1

Report broken media

Score: **21.3%**

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Question 109 of 129

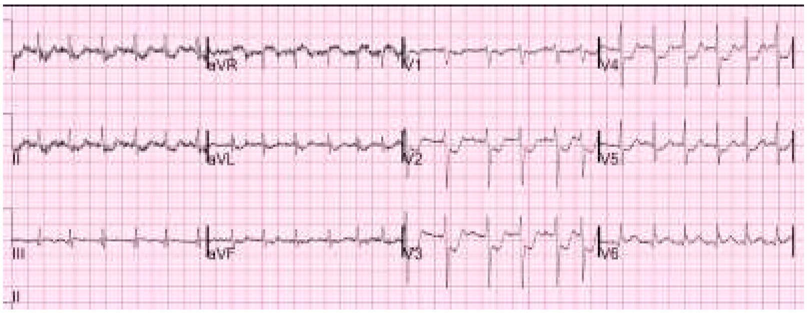
A 34-year-old male is brought in from a nearby bar having collapsed. His friends who accompany him tell you that the patient had been moderately intoxicated but had not reported any symptoms prior to his collapse.

On arrival, the patient is conscious but reports feeling generally weak and light-headed. He has never had an episode like this before but has had episodes of palpitation with associated shortness of breath previously.

The patient states he smokes 20 cigarettes/day, often binge drinks consuming 10 plus pints of beer most weekends and occasionally uses cocaine.

On examination, the patient is conscious and reports some lightheadedness and palpitation but feels otherwise ok when lying down. He has a fast, thready pulse between 130-150bpm and a blood pressure of 89/65mmHg. He has a mildly raised respiratory rate but his chest is clear and peripheral saturations are 94% on room air. Heart sounds are normal and examination is otherwise unremarkable.

An ECG is performed as below.



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What is the most appropriate management?

○ IV adenosine

○ IV metoprolol

○ Cardioversion ×

○ Pacemaker insertion ×

○ Percutaneous coronary intervention ×

Submit answer

Reference ranges ⌵

Score: **21.3%**

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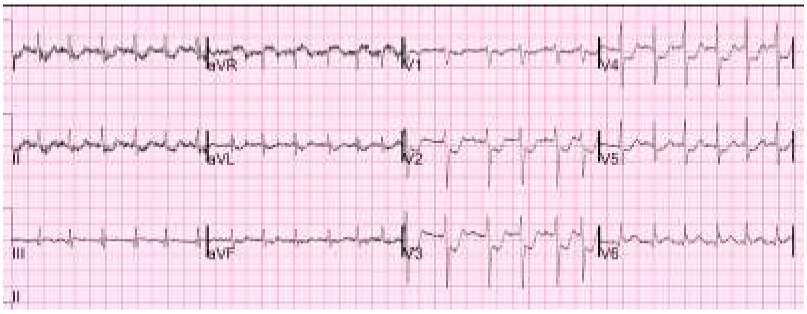

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What is the most appropriate management?

- IV adenosine 13%
- IV metoprolol 5%

Cardioversion	59%
Pacemaker insertion	2%
Percutaneous coronary intervention	22%

This patient has presented with a tachyarrhythmia and is haemodynamically unstable with an acute collapse, most likely syncope induced, and shock, demonstrated by the hypotension and cool extremities, etc. As seen on the ECG the patient's rhythm is fast and irregular with a varied rate of between 100 and 150 bpm. The QRS complexes are narrow and clear 'p' waves cannot be seen before each complex. This is in keeping with a tachyarrhythmia, most likely secondary to atrial fibrillation however the specific cause is not relevant as urgent synchronised cardioversion is required in any unstable patient presenting with a tachyarrhythmia. Cardioversion should be carried out in a resuscitation setting, with procedural sedation if possible. Other indications of instability in arrhythmias include evidence of myocardial ischaemia and heart failure. Following cardioversion, further management is based on the QRS complexes (narrow vs broad) and the rhythm (regular vs irregular).

Adenosine is the treatment of choice in patients presenting with supraventricular tachycardias (SVT), which may be the underlying diagnosis in this case, however, it should be used in hemodynamically stable patients only. As the patient is unstable cardioversion is required.

Metoprolol is an IV beta-blocker and, although it can be used in some tachyarrhythmias, this patient is unstable and requires urgent electrical cardioversion. IV beta-blockers can be used in stable patients with tachyarrhythmias (e.g. atrial fibrillation) for rate control as so long as they have an adequate systolic blood pressure and there are no other contraindications (e.g. asthma).

Pacemaker insertion is used in the management of bradycardia arrhythmias and does not have a role in acute arrhythmias causing compromise.

Percutaneous coronary intervention is used in the management of myocardial infarction (MI). Although the patient has some risk factors, and cocaine use can induce an MI, it is unlikely to be the underlying issue in this case with the patient not reporting chest pain symptoms and no clear evidence of ST-elevation seen on the ECG.

Discuss (10) Improve

Next question

Peri-arrest rhythms: tachycardia

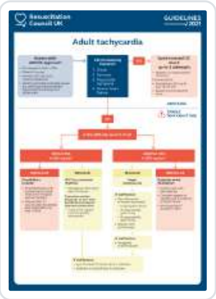
The 2015 Resuscitation Council (UK) guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given. Up to 3 shocks can be given; after this expert help should be sought.

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment guidelines can be found at the Resuscitation Council website, below is a very limited summary:



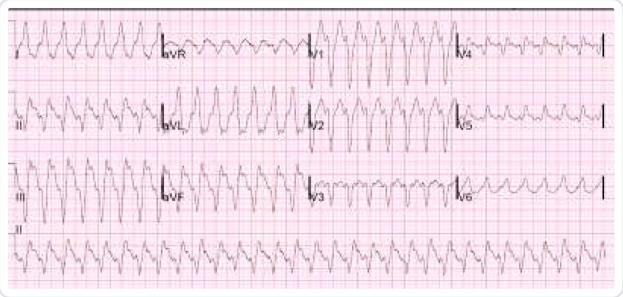
Broad-complex tachycardia

Regular

- assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
- loading dose of amiodarone followed by 24 hour infusion

Irregular

- seek expert help. Possibilities include:
    - atrial fibrillation with bundle branch block - the most likely cause in a stable patient
    - atrial fibrillation with ventricular pre-excitation
    - torsade de pointes



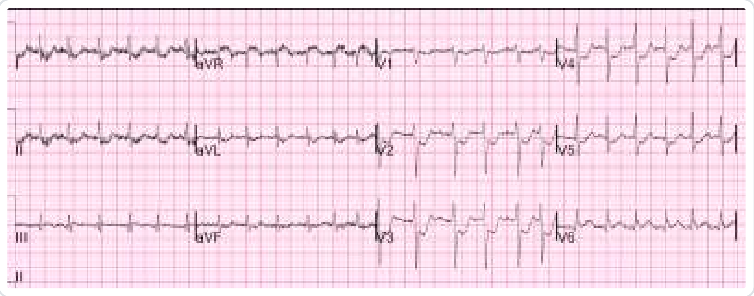
Narrow-complex tachycardia

Regular

- vagal manoeuvres followed by IV adenosine
- if the above is unsuccessful consider a diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

- probable atrial fibrillation
- if onset < 48 hr consider electrical or chemical cardioversion
- rate control: beta-blockers are usually first-line unless there is a contraindication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123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

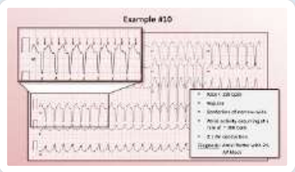
Resuscitation Council UK 👍 30 👎 9

2021 Tachycardia algorithm

Suggest link

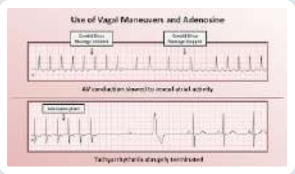
Report broken link

Media



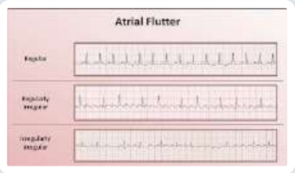
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 5 👎 0


Use of Vagal Maneuvers and Adenosine
AV conduction slowed to reveal atrial activity
Tachyarrhythmia abruptly terminated

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 4 👎 0


Atrial Flutter

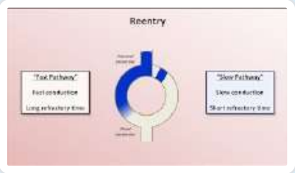
Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 3 👎 1

CARDIOLOGY
ARRHYTHMIA
TACHYCARDIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 1


Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 2 👎 1

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Score: **21.1%**

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109	✗

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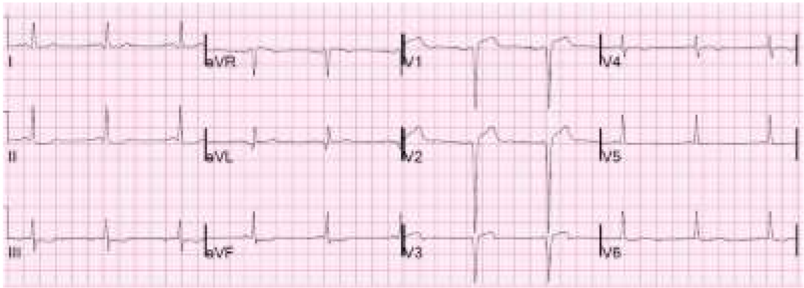
Question 110 of 129

A 55-year-old man presents to the emergency department with a 1-day history of central chest pain. He also complains of progressive shortness of breath on exertion over the last 2 weeks. He was discharged from the cardiology ward 21 days ago after having a drug-eluting stent inserted following an anterolateral myocardial infarction. His past medical history includes diet-controlled type 2 diabetes mellitus and hypertension.

On examination, he is afebrile with a temperature of 36.4°C. His heart rate is 88 beats/min and regular with a blood pressure of 139/82mmHg. On auscultation, there are bibasal crepitations with saturations of 90% on air. There is pitting oedema to his mid-shins with soft and non-tender calves.

An ECG is taken, as shown below:



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What is the most likely diagnosis?

- ○ Anterior myocardial infarction ×
- ○ Left ventricular aneurysm ×
- ○ Papillary muscle rupture ×
- ○ Pericarditis ×
- ○ Pulmonary embolism ×

Submit answer

Question 110 of 129

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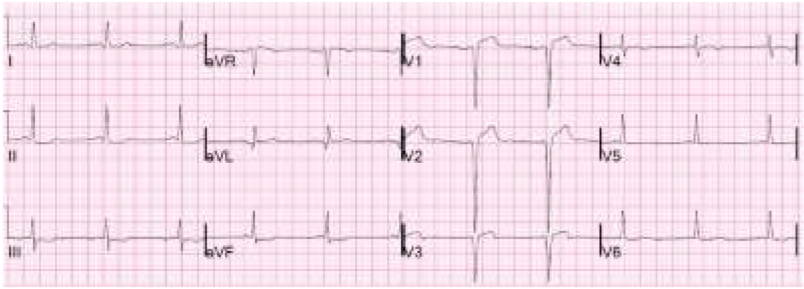

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What is the most likely diagnosis?

Anterior myocardial infarction	17%
Left ventricular aneurysm	59%
Papillary muscle rupture	17%
Pericarditis	5%
Pulmonary embolism	2%

This patient has a diagnosis of a **left ventricular aneurysm**, a rare but serious complication of an anterior myocardial infarction. The complication tends to present more than 2 weeks after a myocardial infarction and symptoms may include chest pain or congestive heart failure (e.g. shortness of breath and fluid overload). This patient's ECG shows ST elevation in V1-V3 with QS waves and upright T waves. Persistent ST elevation after a myocardial infarction is suggestive of a left ventricular aneurysm. However, in a patient with a recent history of ischaemia, it is also important to exclude another ischaemic event. A left ventricular aneurysm is much more likely if there are absent reciprocal ST changes, dynamic ST changes and well-formed Q or QS waves.

Acute coronary syndrome is an important diagnosis to consider following a myocardial infarction. However, ECG findings that are more typical of acute coronary ischaemia include new ST changes with dynamic and reciprocal changes, which are not seen here. Furthermore, the more gradual onset of symptoms is more suggestive of an alternative diagnosis such as a left ventricular aneurysm.

Papillary muscle rupture is incorrect. An anterolateral myocardial infarction causes rupture of the anterolateral papillary muscle, producing symptoms of mitral regurgitation. Although symptoms of congestive cardiac failure are evident in such cases, the onset of symptoms is usually more acute compared to a left ventricular aneurysm. Furthermore, it does not explain this patient's ECG changes or normal heart sounds (where a pansystolic murmur of mitral regurgitation may be seen).

Pericarditis is incorrect. Unlike Dressler's syndrome, post-MI pericarditis is an acute complication within the first 48 hours. The presentation is also similar to acute pericarditis with pleuritic chest pain worse on lying flat.

Pulmonary embolism is incorrect. ECG findings consistent with a pulmonary embolism include sinus tachycardia, RAD, RBBB or S1Q3T3. It does not explain this patient's ST elevation.

👍 👎 💬 Discuss (5) Improve

Next question >

Myocardial infarction: complications ★

Patients are at risk of a number of immediate, early and late complications following a myocardial infarction (MI).

Cardiac arrest

This most commonly occurs due to patients developing ventricular fibrillation and is the most common cause of death following a MI. Patients are managed as per the ALS protocol with defibrillation.

Cardiogenic shock

If a large part of the ventricular myocardium is damaged in the infarction the ejection fraction of the heart may decrease to the point that the patient develops cardiogenic shock. This is difficult to treat. Other causes of cardiogenic shock include the 'mechanical' complications such as left ventricular free wall rupture as listed below. Patients may require inotropic support and/or an intra-aortic balloon pump.

Chronic heart failure

As described above, if the patient survives the acute phase their ventricular myocardium may be dysfunctional resulting in chronic heart failure. Loop diuretics such as furosemide will decrease fluid overload. Both ACE-inhibitors and beta-blockers have been shown to improve the long-term prognosis of patients with chronic heart failure.

Tachyarrhythmias

Ventricular fibrillation, as mentioned above, is the most common cause of death following a MI. Other common arrhythmias including ventricular tachycardia.

Bradyarrhythmias

Atrioventricular block is more common following inferior myocardial infarctions.

Pericarditis

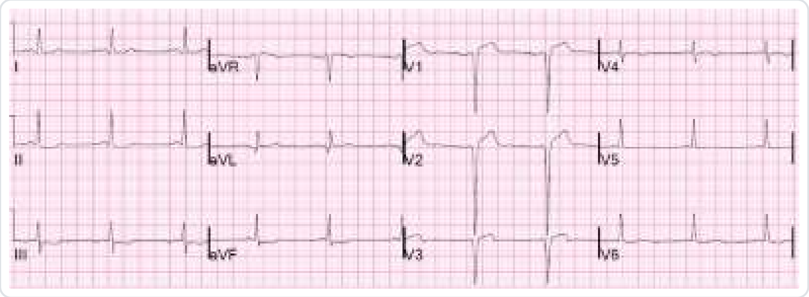
Pericarditis in the first 48 hours following a transmural MI is common (c.

10% of patients). The pain is typical for pericarditis (worse on lying flat etc), a pericardial rub may be heard and a pericardial effusion may be demonstrated with an echocardiogram.

Dressler's syndrome tends to occur around 2-6 weeks following a MI. The underlying pathophysiology is thought to be an autoimmune reaction against antigenic proteins formed as the myocardium recovers. It is characterised by a combination of fever, pleuritic pain, pericardial effusion and a raised ESR. It is treated with NSAIDs.

Left ventricular aneurysm

The ischaemic damage sustained may weaken the myocardium resulting in aneurysm formation. This is typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated.



Left ventricular free wall rupture

This is seen in around 3% of MIs and occurs around 1-2 weeks afterwards. Patients present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). Urgent pericardiocentesis and thoracotomy are required.

Ventricular septal defect

Rupture of the interventricular septum usually occurs in the first week and is seen in around 1-2% of patients. Features: acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed.

Acute mitral regurgitation

More common with infero-posterior infarction and may be due to ischaemia or rupture of the papillary muscle. Acute hypotension and pulmonary oedema may occur. An early-to-mid systolic murmur is typically heard. Patients are treated with vasodilator therapy but often require emergency surgical repair.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 36 👎 22

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Suggest link Report broken link

Score: **20.9%**

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Question 111 of 129

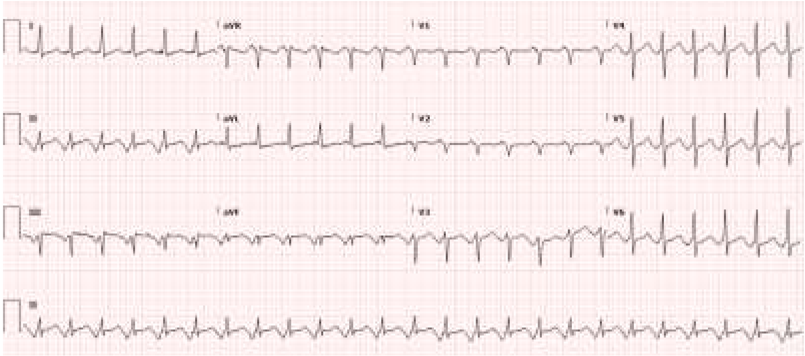
A 26-year-old woman has attended the emergency department with palpitations and shortness of breath. She has a background of asthma for which she has beclometasone and salbutamol inhalers.

On further questioning, she denies chest pain, dizziness, and loss of consciousness.

Observations are as follows:

Heart rate	200 bpm
Blood pressure	102/56 mmHg
Oxygen saturations	98%
Respiratory rate	28 /min
Temperature	36.8ºC

Her ECG is shown below:



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Of the following options, what is the most appropriate management?

○ Direct current (DC) cardioversion ×

○ Intravenous adenosine 12mg ×

○ Intravenous adenosine 6mg ×

○ Intravenous metoprolol ×

○ Intravenous verapamil ×

Submit answer

Reference ranges ∨

Score: **20.9%**

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Question 111 of 129 ✓

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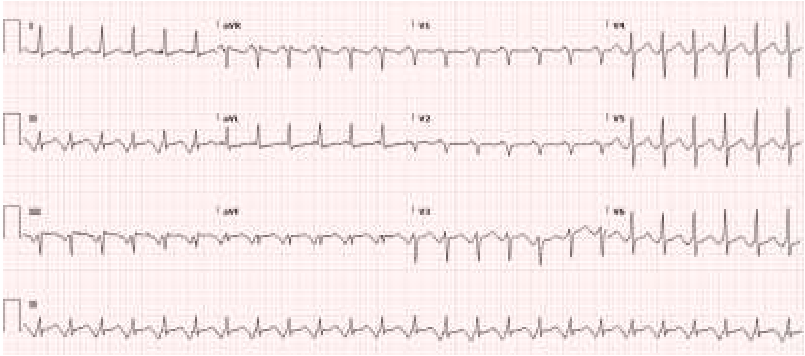

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Of the following options, what is the most appropriate management?

Direct current (DC) cardioversion	12%
Intravenous adenosine 12mg	1%
Intravenous adenosine 6mg	26%
Intravenous metoprolol	2%

Intravenous verapamil 59%

The above ECG shows a narrow complex tachycardia of approximately 200bpm. This is also associated with an absence of P waves, except for P wave inversion in the inferior leads, otherwise known as retrograde conduction. These findings are in keeping with atrioventricular nodal re-entry tachycardia, or AVNRT, the most common form of supraventricular tachycardia.

The correct management option in the above scenario is **intravenous verapamil**. All five of the above options can be used in patients with supraventricular tachycardia, however, verapamil is the most appropriate given the patient's clinical state and past medical history of asthma.

Following advanced life support algorithms, **DC cardioversion** should be used in narrow complex tachyarrhythmias when the patient is unstable. The above scenario does not describe evidence of heart failure, syncope, chest pain, or haemodynamic instability (shock). This option is therefore incorrect.

Intravenous adenosine 12mg is used in chemical cardioversion of narrow complex tachyarrhythmias, however, should only be used when 6mg of adenosine has failed to cardiovert. Furthermore, adenosine is contraindicated in patients with asthma. For these reasons, this option is incorrect.

Intravenous adenosine 6mg is often the initial medical management in narrow complex tachyarrhythmias. Although this would normally be the next option in management, adenosine is contraindicated in patients with asthma, making this option incorrect.

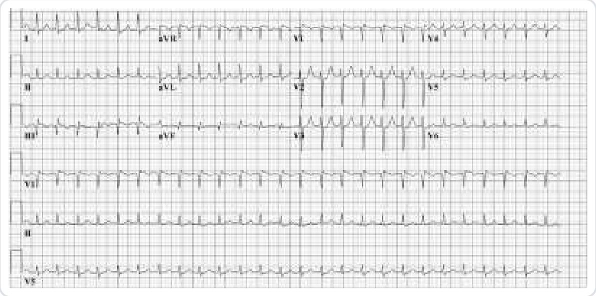
Intravenous metoprolol is also an option in the management of supraventricular tachycardias. Cardioversion is caused by heart rate reduction leading to self-termination of the tachyarrhythmia. However, due to the patient's asthma history, beta-blockers should be avoided.

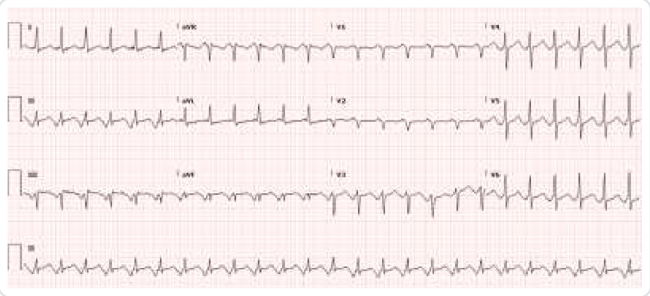
Discuss (12) | Improve

Next question ›

Supraventricular tachycardia ★

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
  - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
  - carotid sinus massage
- intravenous adenosine
  - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
  - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

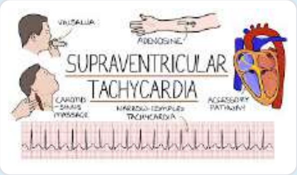
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

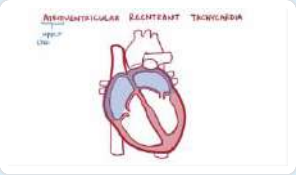
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

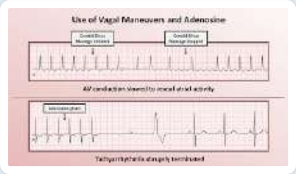
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0



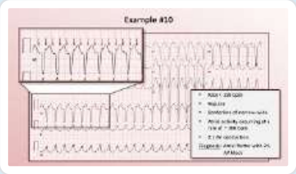
Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0


Use of Vagal Maneuvers and Adenosine

Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0



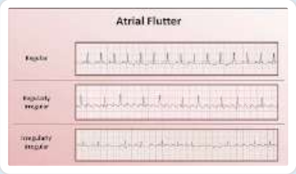
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0

AVRT & AVNRT
Khan Academy

Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0


Atrial Flutter

Intro to EKG Interpretation - Overview of Tachyarrhythmias

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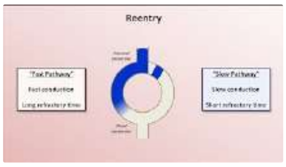
Reentry
"Fast Pathway"
Fast conduction
Long refractory time
"Slow Pathway"
Slow conduction
Short refractory time

Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube

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Question 112 of 129

A 55-year-old Afro-Caribbean man visits his general practitioner for a follow-up of his hypertension management. He was diagnosed with hypertension 5 years ago, and his past medical history is significant for hypercholesterolemia. His hypertension has been treated with ramipril and amlodipine. Over the past 6 months, his blood pressure chart shows consistently high readings. He has already tried decreasing salt intake and exercising more. He does not smoke or consume alcohol.

On examination, his blood pressure is 156/97 mmHg, and pulse is 80/min. His BMI is 32 kg/m^2 with an increased waist-to-hip ratio.

Which of the following is the next best step in the management of this patient's hypertension?

- ○ Add benazepril
- ○ Add chlortalidone
- ○ Change ramipril to losartan
- ○ Refer to bariatric surgery
- ○ Advise lifestyle changes and reassess in 6 months

Submit answer

Reference ranges

Score: **21.6%**

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111	✓
112	-

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Question 112 of 129 ✗

A 55-year-old Afro-Caribbean man visits his general practitioner for a follow-up of his hypertension management. He was diagnosed with hypertension 5 years ago, and his past medical history is significant for hypercholesterolemia. His hypertension has been treated with ramipril and amlodipine. Over the past 6 months, his blood pressure chart shows consistently high readings. He has already tried decreasing salt intake and exercising more. He does not smoke or consume alcohol.

On examination, his blood pressure is 156/97 mmHg, and pulse is 80/min. His BMI is 32 kg/m^2 with an increased waist-to-hip ratio.

Which of the following is the next best step in the management of this patient's hypertension?

Add benazepril	2%
Add chlortalidone	63%
Change ramipril to losartan	21%
Refer to bariatric surgery	6%
Advise lifestyle changes and reassess in 6 months	7%

In black individuals who are already taking a calcium channel blocker and ACE-inhibitor, and who continue to have blood pressure that is difficult to control, adding a thiazide-like diuretic is the next most appropriate step

Important for me Less important

This patient has primary hypertension that is not controlled by an ACE-inhibitor and calcium channel blocker. The addition of another antihypertensive drug is the most appropriate management in this case. The choice of drug depends on the race of the patient. For black individuals, the recommendation is to add a thiazide-like diuretic, like chlortalidone or indapamide.

This patient is already taking an ACE inhibitor, ramipril. The guidelines recommend adding a drug with a different mechanism of action. Adding another ACE inhibitor, like benazepril, is inappropriate.

In some patients, ACE inhibitors may cause side effects. In such patients, it is suggested to replace the ACE-inhibitor with angiotensin-receptor blockers, like losartan. This patient does not report any side effects associated with ACE inhibitors.

Bariatric surgery is a treatment option for people with obesity if all of the following criteria are fulfilled:

- A BMI of 40 kg/m^2 or more, or
- BMI between 35 kg/m^2 and 40 kg/m^2 and another significant disease (for example, type 2 diabetes or high blood pressure) that could be improved if they lost weight.

With a BMI of 32 kg/m^2, this patient does not fulfil the criteria for bariatric surgery.

This patient has already tried lifestyle changes. Although he should continue these lifestyle changes, he also needs a change in his drug regimen.

👍 👎 Discuss (8) Improve

Next question >

Thiazide diuretics ★

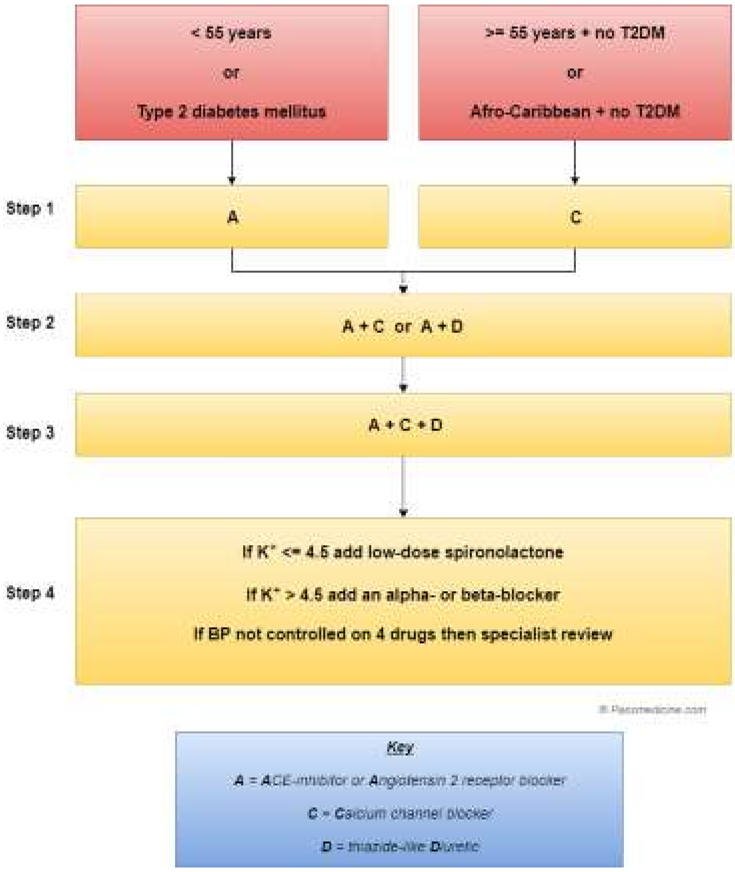
Thiazide diuretics work by inhibiting sodium reabsorption at the beginning of the distal convoluted tubule (DCT) by blocking the thiazide-sensitive Na^{+}-Cl$^{\text{ˆ’}}$ symporter. Potassium is lost as a result of more sodium reaching the collecting ducts. Thiazide diuretics have a role in the treatment of mild heart failure although loop diuretics are better for reducing overload. The main use of bendroflumethiazide was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as indapamide and chlortalidone.

Common adverse effects

- dehydration
- postural hypotension
- hypokalaemia
    - due to increased delivery of sodium to the distal part of the distal convoluted tubule → increased sodium reabsorption in exchange for potassium and hydrogen ions
- hyponatraemia
- hypercalcaemia
    - the flip side of this is hypocalciuria, which may be useful in reducing the incidence of renal stones
- gout
- impaired glucose tolerance
- impotence

Rare adverse effects

- thrombocytopaenia
- agranulocytosis
- photosensitivity rash
- pancreatitis


< 55 years
or
Type 2 diabetes mellitus
>= 55 years + no T2DM
or
Afro-Caribbean + no T2DM
Step 1
A
C
Step 2
A + C or A + D
Step 3
A + C + D
Step 4
If K⁺ <= 4.5 add low-dose spironolactone
If K⁺ > 4.5 add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
© Passmedicine.com
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

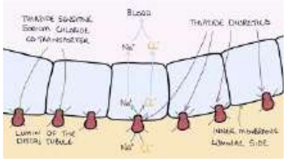
High-yield textbook

Extended textbook

Media

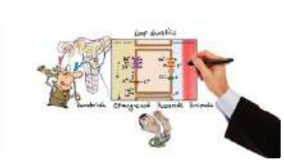
Pharmacology - Diuretics

Armando Hasudungan - YouTube 5 0



How do Thiazide Diuretics Work? Understanding Bendroflumethiazide and Indapamide

Zero To Finals - YouTube 4 0



Diuretics

Speed Pharmacology - YouTube 👍 2 👎 0

Report broken media

Score: **21.4%**

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111	✓
112	✗

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A 32-year-old woman presents to the emergency department with palpitations. Her past medical history includes type 2 diabetes mellitus and acne vulgaris. She is currently taking Yasmin (a combined oral contraceptive pill) and has no known allergies.

Examination reveals a strong radial pulse, which is tachycardic and regular. She has a raised body mass index. Her cardiopulmonary system is unremarkable.

Observations:

- Heart rate: 168/min
- Blood pressure: 138/88 mmHg
- Oxygen saturations: 98% on room air
- Temperature: 37ºC

An ECG shows a supraventricular tachycardia.

The foundation year two doctor has already tried vagal manoeuvres and has just given 6mg of adenosine to no effect.

What is the most appropriate next step in management?

- ○ Amiodarone IV infusion
- ○ 12mg adenosine IV
- ○ Beta blocker IV
- ○ Cardioversion
- ○ Further 6mg adenosine IV

Submit answer

Reference ranges

Score: **21.4%**

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Back to top

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Examination reveals a strong radial pulse, which is tachycardic and regular. She has a raised body mass index. Her cardiopulmonary system is unremarkable.

Observations:

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- Blood pressure: 138/88 mmHg
- Oxygen saturations: 98% on room air
- Temperature: 37ºC

An ECG shows a supraventricular tachycardia.

The foundation year two doctor has already tried vagal manoeuvres and has just given 6mg of adenosine to no effect.

What is the most appropriate next step in management?

Amiodarone IV infusion	1%
12mg adenosine IV	85%
Beta blocker IV	1%
Cardioversion	1%
Further 6mg adenosine IV	13%

Supraventricular tachycardia follows the narrow complex, regular tachycardia ALS guidelines.

If the patient is haemodynamically stable with no adverse features, such as shock, myocardial ischaemia or heart failure:

- 1. Start with vagal manoeuvres. Carotid sinus massage or the Valsalva manoeuvre
- 2. Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg
- 3. If unsuccessful, seek specialist help

Use a large cannula to administer the adenosine and give a rapid bolus. A history of asthma is an important contraindication to adenosine, as it can cause bronchospasm. Instead, a calcium channel blocker should be used.

If the patient in unstable, they should the electrically cardioverted start with 70-120 J biphasic (100 J monophasic).

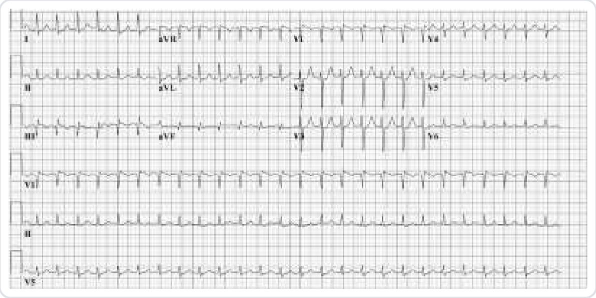
Reference: ALS peri-arrest algorithm
Found at : https://www.resus.org.uk/pages/periarst.pdf

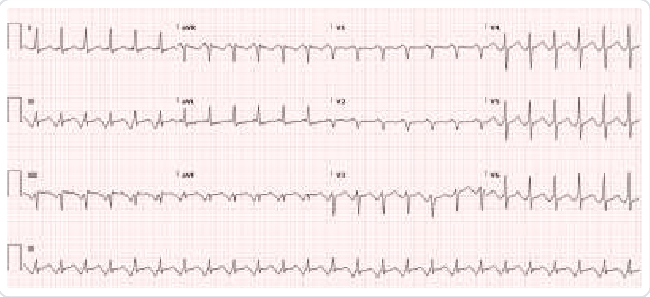
👍 👎 Discuss (5) Improve

Next question >

Supraventricular tachycardia

Whilst strictly speaking the term supraventricular tachycardia (SVT) refers to any tachycardia that is not ventricular in origin the term is generally used in the context of paroxysmal SVT. Episodes are characterised by the sudden onset of a narrow complex tachycardia, typically an atrioventricular nodal re-entry tachycardia (AVNRT). Other causes include atrioventricular re-entry tachycardias (AVRT) and junctional tachycardias.





Management

Acute management

- vagal manoeuvres:
  - Valsalva manoeuvre: e.g. trying to blow into an empty plastic syringe
  - carotid sinus massage
- intravenous adenosine
  - rapid IV bolus of 6mg → if unsuccessful give 12 mg → if unsuccessful give further 18 mg
  - contraindicated in asthmatics - verapamil is a preferable option
- electrical cardioversion

Prevention of episodes

- beta-blockers
- radio-frequency ablation

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Resuscitation Council UK 👍 25 👎 8

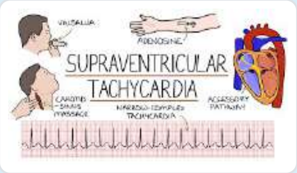
2021 Tachycardia algorithm

Life in the Fast Lane 👍 24 👎 6

Supraventricular tachycardia

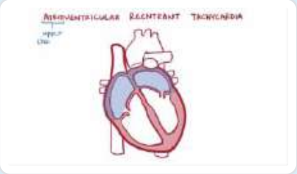
Suggest link Report broken link

Media


VALSALVA
ADENOSINE
SUPRAVENTRICULAR TACHYCARDIA
CAROTID SINUS MASSAGE
NARROW COMPLEX TACHYCARDIA
ACCESSORY PATHWAY

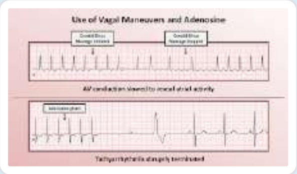
Understanding Supraventricular Tachycardia (SVT)

Zero To Finals - YouTube 👍 19 👎 0


ATRIOVENTRICULAR REENTRANT TACHYCARDIA

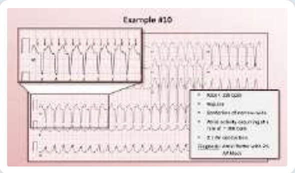
Atrioventricular reentrant tachycardia (AVRT) & nodal reentrant tachycardia (AVNRT)

Osmosis - YouTube 👍 8 👎 0



Intro to EKG Interpretation - How to Identify Any Tachyarrhythmia with 6 Easy Questions

Strong Medicine - YouTube 👍 5 👎 0



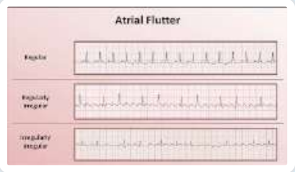
Intro to EKG Interpretation - Practicing Tachyarrhythmia Identification

Strong Medicine - YouTube 👍 2 👎 0

AVRT & AVNRT
Khan Academy

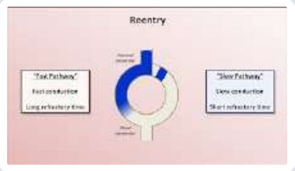
Atrioventricular reentrant tachycardia (AVRT) & AV nodal reentrant tachycardia (AVNRT)

Khan Academy - YouTube 👍 2 👎 0


Atrial Flutter

Intro to EKG Interpretation - Overview of Tachyarrhythmias

Strong Medicine - YouTube 👍 0 👎 0



Intro to EKG Interpretation - Mechanisms of Tachyarrhythmias

Strong Medicine - YouTube 👍 1 👎 1

Report broken media

Score: **22.1%**

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113 ✓

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Question 114 of 129

A 72-year-old man presents to the emergency department with dizziness. There is no chest pain, palpitations or shortness of breath. He has a past medical history of hypertension. He takes amlodipine. He does not smoke or drink alcohol.

His observations are as follows:

- Heart rate 48 beats per minute
- Blood pressure 125/84 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

On examination, he is now asymptomatic. His heart rate is irregular. There are no murmurs. Chest auscultation is normal. His jugular venous pulse is not elevated. His Glasgow coma scale is 15/15.

An ECG demonstrates intermittently non-conducted P waves associated with a prolonged PR interval that is constant and not progressive. The QRS is broad. There are no ischaemic changes.

Bedside echocardiography reveals an approximately normal ejection fraction.

Given the likely diagnosis, what is the most appropriate management?

○ Atropine

○ Biventricular implantable cardioverter defibrillator (ICD) insertion

○ Dual chamber pacemaker insertion

○ Monitoring

○ Single chamber pacemaker insertion

Submit answer

Reference ranges ∨

Score: **22.1%**

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114	-

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Bedside echocardiography reveals an approximately normal ejection fraction.

Given the likely diagnosis, what is the most appropriate management?

Atropine	6%
Biventricular implantable cardioverter defibrillator (ICD) insertion	8%
Dual chamber pacemaker insertion	62%
Monitoring	12%
Single chamber pacemaker insertion	12%

In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated

Important for me Less important

Dual chamber pacemaker insertion is the correct answer. The patient has high grade atrioventricular (AV) block (2nd degree, Mobitz type II). This is characterised by intermittent non-conduction of P waves associated with a prolonged PR interval. In contrast to Mobitz type I, the PR interval is constantly prolonged and does not progressively lengthen. Mobitz type II is typically caused by significant structural damage to the conducting system. Mobitz II is much more likely than Mobitz I to degenerate to haemodynamic compromise and progress to complete heart block. Pacemaker insertion is warranted. It should be a dual chamber pacemaker on setting DDD or DDDR. DDD pacing means that the pacemaker can record both atrial and ventricular rates and can pace either chamber as necessary. DDDR is the same but can adjust the heart rate to account for the need for higher cardiac output.

Atropine is incorrect. 2nd degree AV block is typically not responsive to atropine. Additionally, while this patient certainly needs a pacemaker, he is now asymptomatic with no adverse features and therefore does not need atropine urgently.

Biventricular implantable cardioverter defibrillator (ICD) insertion is incorrect. This is typically indicated in patients with an ejection fraction of < 35% and symptomatic heart failure. It is not strictly inserted for high risk bradyarrhythmias.

Monitoring is incorrect. He is at risk of sudden deterioration and needs definitive management.

Single chamber pacemaker insertion is incorrect. Single chamber pacemakers are predominantly used for the management of atrial arrhythmias and are not indicated in those with AV block.

Discuss (8) Improve

Next question

Pacemaker: permanent

A permanent pacemaker (PPM) is an electronic device that maintains a consistent and physiologically adequate heart rate when the natural cardiac pacemaker system fails.

Background Anatomy/Physiology

Electrical impulses originate from the sinoatrial (SA) node in the right atrium, leading to atrial contraction. The impulse then travels to the atrioventricular (AV) node and subsequently along the Purkinje fibres, culminating in ventricular contraction. This coordinated activity results in an effective cardiac output.

Dysfunction at any level of this conduction system can result in arrhythmias. A PPM, once implanted, helps to rectify these abnormalities by delivering timed electrical stimuli to the myocardium, promoting effective contractions and thereby maintaining circulation.

Indications

Indications for permanent pacemaker (PPM) insertion include:

- persistent symptomatic bradycardia e.g. sick sinus syndrome
- complete heart block
- Mobitz type II AV block
- persistent AV block after myocardial infarction

Classification and types

Nomenclature of pacemakers

There is a standard code to describe the functions of a pacemaker in a systematic and concise manner. Each position in the code denotes a specific characteristic of the pacemaker's function. The code typically consists of three to five positions.

First Position (Chamber Paced):

- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)

- O - None (no pacing)

Second Position (Chamber Sensed):
- V - Ventricle
- A - Atrium
- D - Dual (both atrium and ventricle)
- O - None (no sensing)

Third Position (Response to Sensing):
- I - Inhibited (pacemaker is prevented from pacing if it senses intrinsic cardiac activity)
- T - Triggered (pacemaker will release a pulse if it senses intrinsic activity, usually used in combination therapies)
- D - Dual (both inhibited and triggered)
- O - None (no response to sensing)

For pacemakers with more advanced functionalities, there are often fourth and fifth positions:

Fourth Position (Rate Modulation):
- R - Rate modulated (the pacemaker can adjust its pacing rate in response to changes in the patient's activity or other physiological indicators)
- O - None (no rate modulation)

Fifth Position (Multi-Site Pacing):
- A - Atrium
- V - Ventricle
- D - Dual (both atrium and ventricle)
- O - None (no multi-site pacing)

Examples:
- VVI: A pacemaker that paces the ventricle, senses the ventricle, and will be inhibited if it detects an intrinsic ventricular beat.
- AAI: A pacemaker that paces the atrium, senses the atrium, and will be inhibited if it detects an intrinsic atrial beat.
- DDD: A pacemaker that paces and senses in both the atrium and ventricle. It can both inhibit and trigger pacing based on its sensing.
- VVIR: A ventricular paced, ventricular sensed, inhibited response pacemaker that can adjust its rate based on physiologic demand.

Types of pacemaker

Permanent pacemakers come in various configurations based on their lead placement, the number of chambers they interact with, and their specific functionalities.

Single-chamber pacemakers:

- Ventricular (VVI) pacemaker: This is the most common type of single-chamber pacemaker. The lead is placed in the ventricle (typically the right ventricle), and it both senses and paces in that chamber. It is particularly useful for patients with atrial fibrillation and AV block.
- Atrial (AAI) pacemaker: Less common than VVI, this pacemaker's lead is placed in the atrium (usually the right atrium). It senses and paces the atrium and is primarily used in patients with sinus node dysfunction without AV block.

Dual-chamber pacemakers (DDD):

- This type places leads in both the right atrium and right ventricle. It can sense and pace in both chambers, allowing for more physiological pacing by preserving the natural sequence of atrial and ventricular contraction. This is particularly beneficial for patients with sinus node dysfunction and AV block.

Rate-responsive pacemakers:

- These pacemakers adjust the pacing rate based on physiological demand. They use various sensors, such as those detecting motion or respiratory rate, to gauge the patient's activity level and adjust pacing accordingly. Both single-chamber (AAIR or VVIR) and dual-chamber (DDDR) pacemakers can be rate-responsive.

Biventricular pacemakers/Cardiac Resynchronization Therapy (CRT) devices:

- These are specialised devices used primarily for heart failure patients with ventricular dyssynchrony. In addition to the standard right atrium and right ventricle leads, they have a lead placed on the outer surface of the left ventricle, allowing for simultaneous pacing of both ventricles. This can enhance cardiac performance in selected patients by ensuring that the two ventricles contract synchronously.

- Some CRT devices also incorporate defibrillation capabilities (CRT-D) and can deliver shocks if a life-threatening ventricular arrhythmia is detected.

+ Q 123

Next question >

Textbooks

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Media

DUAL CHAMBER
PACEMAKER
RIGHT ATRIUM
AND
RIGHT VENTRICLE
SYNCHRONISE

Pacemakers

Zero To Finals - YouTube 👍 112 👎 2

Report broken media

Score: **21.9%**

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112 ✗

113 ✓

114 ✗

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Question 115 of 129

A 58-year-old woman was referred to the acute medical unit with headaches and hypertension. She had no history of fever, neck stiffness, limb weakness, seizures or blurring of her vision. Her pulse rate was 70 beats per minute and blood pressure was 200/110 mmHg. Her heart sounds, chest examination, abdominal and neurological examination were normal. Fundoscopy was also normal.

Hb	138g/l
Platelets	238 * 10^9/l
WBC	6.2 * 10^9/l
Na^+	135 mmol/l
K^+	3.8 mmol/l
Urea	6.4 mmol/l
Creatinine	75 µmol/l
ECG	normal sinus rhythm
Chest x-ray	normal
CT Head	normal
Urinalysis	normal

What is the best initial management?

- ○ Intravenous labetalol ×
- ○ Intravenous glyceryl trinitrate ×
- ○ Oral amlodipine ×
- ○ Intravenous sodium nitroprusside ×
- ○ Sublingual nifedipine ×

Submit answer

A 58-year-old woman was referred to the acute medical unit with headaches and hypertension. She had no history of fever, neck stiffness, limb weakness, seizures or blurring of her vision. Her pulse rate was 70 beats per minute and blood pressure was 200/110 mmHg. Her heart sounds, chest examination, abdominal and neurological examination were normal. Fundoscopy was also normal.

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ECG	normal sinus rhythm
Chest x-ray	normal
CT Head	normal
Urinalysis	normal

What is the best initial management?

Intravenous labetalol	37%
Intravenous glyceryl trinitrate	4%
🟩 Oral amlodipine	46%
Intravenous sodium nitroprusside	9%
🟥 Sublingual nifedipine	4%

In an hypertensive urgency, treatment aims to lower blood pressure with the use of oral anti-hypertensive medication like a calcium channel blocker

Important for me Less important

This woman has hypertensive urgency, which is a severe increase in blood pressure (>180 mmHg systolic or >110 mmHg diastolic) not associated with target organ damage. This may be associated with headache, shortness of breath, or nosebleeds. The treatment is to lower the blood pressure within 24-48 hours and oral antihypertensive medications are recommended. Hospitalisation is not required. Amlodipine is a dihydropyridine calcium channel antagonist and a potent antihypertensive that can be used in this situation.

Intravenous antihypertensives like labetalol, glyceryltrinitrate are used in hypertensive emergencies which require lowering of blood pressure within minutes to hours e.g hypertensive encephalopathy, aortic dissection. Sublingual nifedipine is not recommended for treatment of hypertensive crises.

👍 👎 Discuss (10) Improve

Next question >

Hypertension: management ★

NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

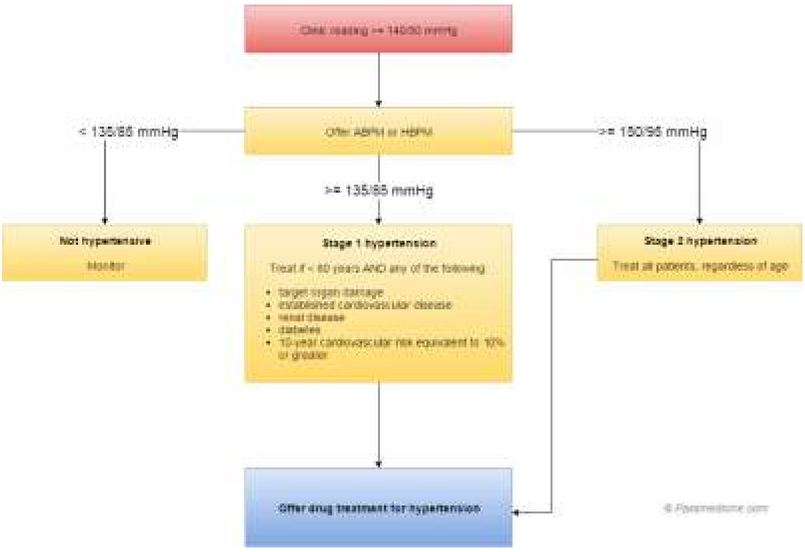
- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that

NICE advocates.

Stage	**Criteria**
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
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Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

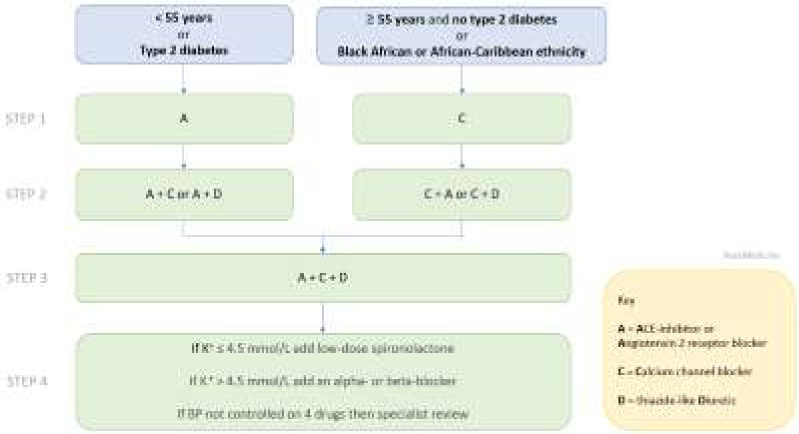
- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced

- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)
- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)
- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment
- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**

    - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
    - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
    - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

- add a third drug to make, i.e.:
    - if already taking an **(A + C)** then add a **D**
    - if already **(A + D)** then add a **C**
- **(A + C + D)**

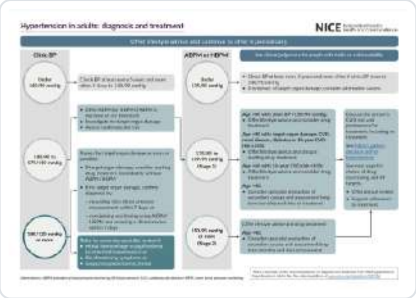
Step 4 treatment

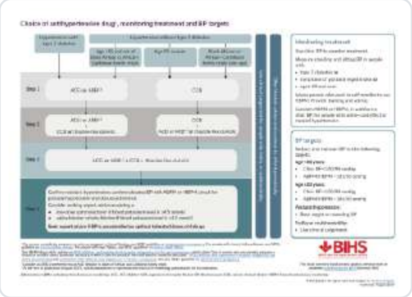
- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
    - confirm elevated clinic BP with ABPM or HBPM
    - assess for postural hypotension.
    - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or*

> *maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*


Hypertension in adults: diagnosis and treatment
NICE


Choice of antihypertensive drug, monitoring treatment and BP targets
BIHS

Blood pressure targets

	Clinic BP	ABPM / HBPM
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

123

Next question

Textbooks

High-yield textbook

Extended textbook

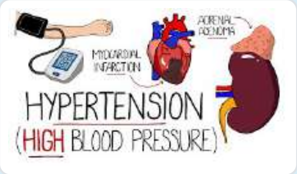
Links

NICE 👍 162 👎 162

2019 Hypertension guidelines

Suggest link Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

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Score: **21.7%**

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Question 116 of 129

A 72-year-old female presents with a 6-month history of gradual onset, progressive exertional dyspnoea associated bilateral increased lower limb pitting oedema. She has no previous cardiac history and prior to 6 months ago, had no limitations to exercise tolerance. Her past medical history includes B cell lymphoma diagnosed four years ago and in remission one course of chemotherapy. On examination, the jugular venous pulse is raised at 6cm above the angle of Louis. A soft systolic murmur and bibasal inspiratory crackles can be heard on auscultation. Abdominal examination demonstrates a pulsatile 3cm liver edge. Her blood pressure is 125/77 mmHg, heart rate 68/min and sinus, saturations 92% on 2l, respiratory rate 22/min. ECG reveals sinus rhythm of low voltage. Chest radiography demonstrates bibasal alveolar shadowing with prominent upper lobe vasculature.

Urine dip is as follows: blood -ve, leucocytes -ve, nitrates -ve protein 3+ pH 5.5.

Blood tests are as follows:

Hb	111 g/l
Platelets	275 * 10^9/l
WBC	8.3 * 10^9/l

Na^+	139 mmol/l
K^+	4.9 mmol/l
Urea	7.4 mmol/l
Creatinine	140 µmol/l
CRP	3 mg/l
Trop	<0.03 (normal range < 0.03)

Bilirubin	6 µmol/l
ALP	32 u/l
ALT	260 u/l

A transthoracic echocardiogram is performed, reported as significant increase in echogenicity, symmetrical left ventricular wall thickening, impaired bilateral diastolic ventricular dysfunction, bilaterally enlarged atria, pulmonary artery systolic pressure 45 mmHg, a 5mm pericardial effusion

What is the most likely underlying diagnosis for this patient?

- ○ Amyloid cardiomyopathy ×
- ○ Non-ST elevation myocardial infarction (NSTEMI) ×
- ○ Idiopathic dilated cardiomyopathy ×
- ○ Chemotherapy induced cardiomyopathy ×
- ○ Viral pericarditis leading to tamponade ×

Submit answer

Reference ranges

Score: **21.7%**

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What is the most likely underlying diagnosis for this patient?

Amyloid cardiomyopathy	53%
Non-ST elevation myocardial infarction (NSTEMI)	1%
Idiopathic dilated cardiomyopathy	3%
Chemotherapy induced cardiomyopathy	41%
Viral pericarditis leading to tamponade	2%

A clinical picture of decompensated congestive cardiac failure is described with subtle details of hepatomegaly (possible congested, possibly related to the underlying diagnosis), associated heavy proteinuria, echogenic TTE and low voltage criteria ECG, on a background of previous B-cell lymphoma. The unifying diagnosis is amyloid cardiomyopathy, likely secondary to AL amyloid from B-cell dyscrasia, with cardiac, renal and possibly liver involvement. There is no evidence of a sudden onset cardiac event suggestive of an ischaemic cause. Echocardiogram does not demonstrate ventricular dilation, only impaired function. A 5mm pericardial effusion is not sufficiently significant to cause tamponade. Although chemotherapy-induced cardiomyopathy is possible, it is difficult to tie in the delayed onset from the end of treatment with renal involvement. It also does not explain the echogenic pictures on transthoracic echo.

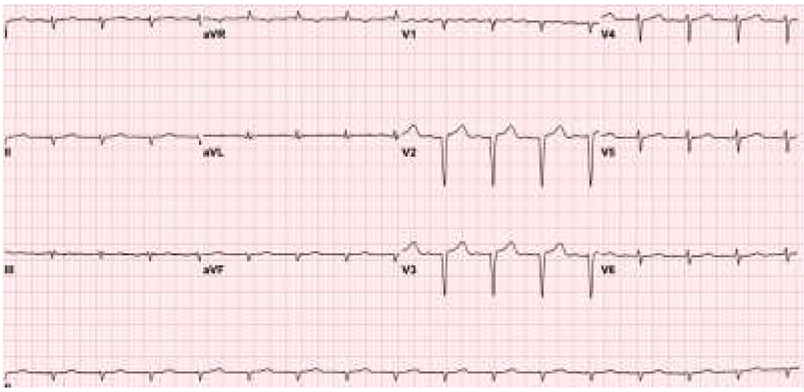
👍 👎 Discuss (8) Improve

Next question ›

Amyloidosis: cardiac ★

Cardiac amyloidosis, also known as amyloid cardiomyopathy, is a rare disorder characterized by the extracellular deposition of insoluble amyloid fibrils in the myocardium, leading to progressive heart failure and conduction abnormalities. Cardiac amyloidosis can occur in isolation or as part of systemic amyloidosis, which affects multiple organs.

Investigations
- ECG: typically shows low-voltage complexes with poor R wave progression in the chest leads (a pseudoinfarction pattern).
- echocardiogram: increased myocardial echogenicity (granular sparkling or 'global speckled' appearance)



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123

Next question

Textbooks

High-yield textbook

Extended textbook

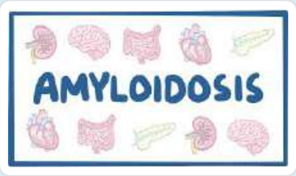
Links

Journal of the American Heart Association 👍 6 👎 0

Updates in Cardiac Amyloidosis: A Review

Suggest link Report broken link

Media



Amyloidosis

Osmosis - YouTube 👍 17 👎 2

Report broken media

Score: **21.6%**

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Question 117 of 129

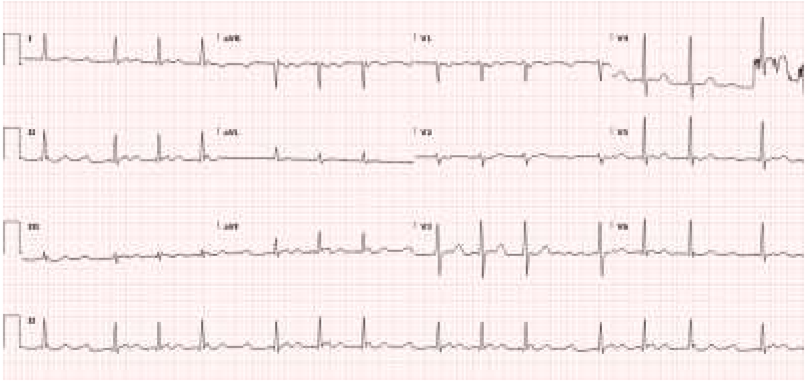
A 68-year-old man presents with occasional lightheadedness over the past few weeks. During his morning walk today, he experienced a brief spell of dizziness. He denies experiencing chest pain, breathlessness, or any other significant complaints.

His medical history includes diabetes, hypertension, and myocardial infarction for which he underwent coronary artery bypass grafting (CABG) seven years ago. He is currently on medications including aspirin, bisoprolol, lisinopril, atorvastatin, and vildagliptin.

Observations are as below:

- BP 140/85 mmHg
- Heart rate 74 bpm
- Respiratory rate 16/min
- Oxygen saturation 98% on room air

An ECG was subsequently carried out.



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What is the most appropriate management option?

○ Discharge him and recommend a follow up ECG ×

○ Give atropine ×

○ Intravenous infusion of isoproterenol ×

○ Refer for cardiac resynchronisation therapy ×

○ Review his medications ×

Submit answer

Reference ranges ∨

Score: **21.6%**

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Question 117 of 129

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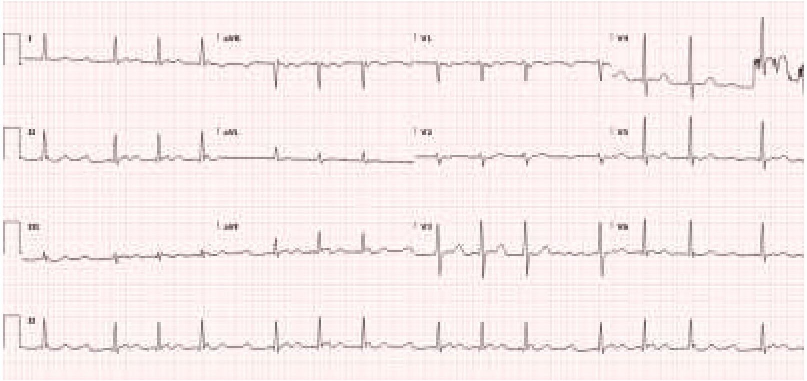

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What is the most appropriate management option?

- Discharge him and recommend a follow up ECG 12%
- Give atropine 5%
- Intravenous infusion of isoproterenol 6%

Refer for cardiac resynchronisation therapy 37%

Review his medications 41%

The correct answer is **review his medications**.

The ECG findings demonstrate a progressive prolongation of the PR interval until a beat is dropped. This is characteristic of a 2nd-degree Mobitz 1 or Wenckebach-type block. This is usually a relatively benign rhythm with a low risk of progression to higher-degree blocks. This patient has symptoms of lightheadedness and dizziness, coupled with stable vital signs. As long as there is no hemodynamic compromise, there is no need for urgent intervention. Searching for a reversible cause should be the first step. This patient is on bisoprolol, a known cause of AVN block, and should be reviewed.

Discharge him and recommend a follow up ECG is incorrect. It is indicated if the patient is asymptomatic with a first or 2nd-degree Mobitz 1 heart block. This patient has symptoms of light-headedness and dizziness, which require intervention.

Give atropine is incorrect. Atropine is generally indicated for acute symptomatic AVN block with bradycardia and adverse features (heart failure, shock, syncope, myocardial ischaemia). There are no adverse features in this case.

Intravenous infusion of isoproterenol is also an incorrect answer. Reviewing any potential causes should be the initial approach for symptomatic hemodynamically stable patients. IV isoproterenol is indicated for hemodynamically unstable patients who fail to respond to transcutaneous pacing. Given the patient's history of coronary artery disease, it is important to avoid it if possible, due to the potential development of ischemic chest pain.

Refer for cardiac resynchronisation therapy is incorrect. Cardiac resynchronisation can be beneficial if there is heart failure with an ejection fraction of less than or equal to 35% and a widened QRS (e.g. left bundle branch block) complex on the ECG. It has no role in the management of the AVN block.

👍 👎 Discuss (4) Improve

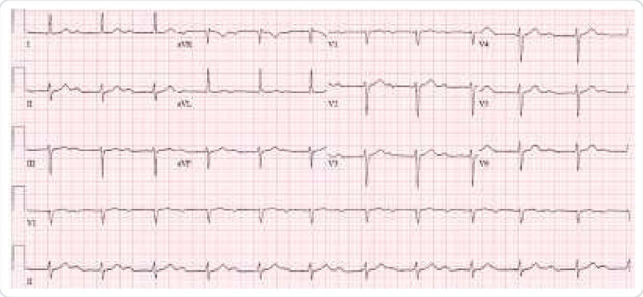
Next question >

Atrioventricular block

In atrioventricular (AV) block, or heart block, there is impaired electrical conduction between the atria and ventricles. There are three types:

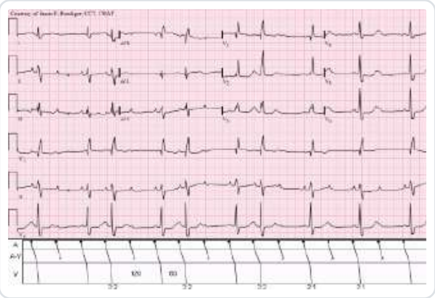
First-degree heart block

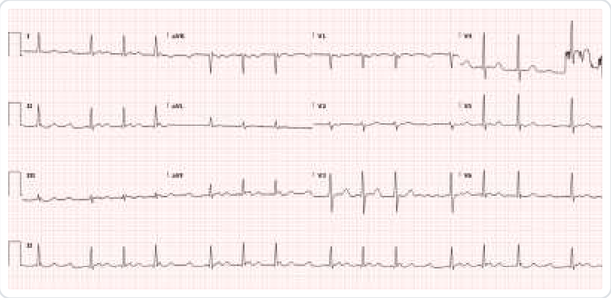
- PR interval > 0.2 seconds
- asymptomatic first-degree heart block is relatively common and does not need treatment



Second-degree heart block

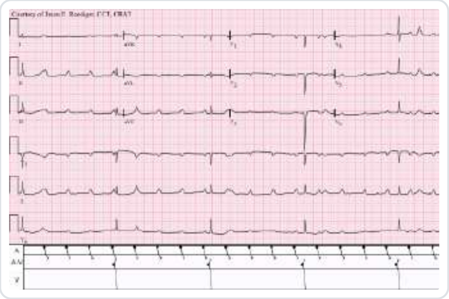
- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex

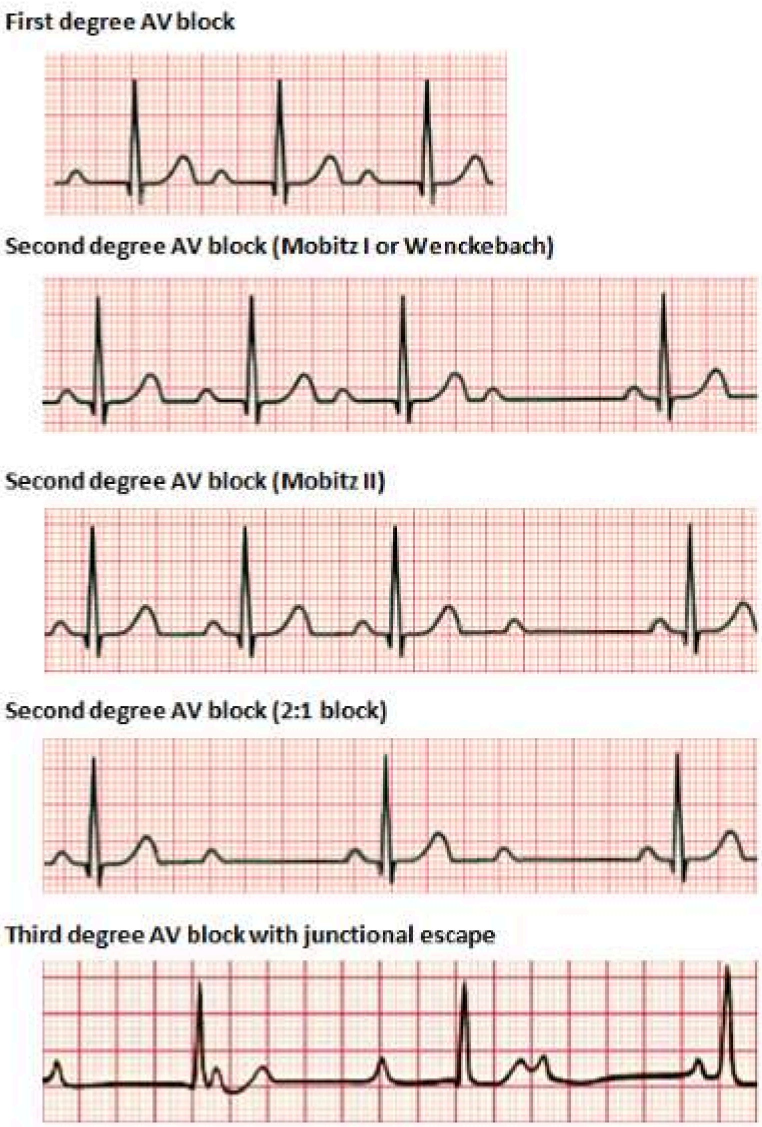




Third-degree (complete) heart block

- there is no association between the P waves and QRS complexes




First degree AV block

Second degree AV block (Mobitz I or Wenckebach)

Second degree AV block (Mobitz II)

Second degree AV block (2:1 block)

Third degree AV block with junctional escape

ECG showing types of heart block

Next question ›

Textbooks

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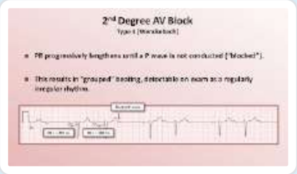
Extended textbook

Media

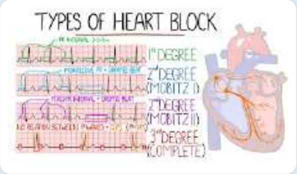

2nd Degree AV Block

AV block

Strong Medicine - YouTube 👍 7 👎 0

Atrioventricular block

Osmosis - YouTube 👍 1 👎 0


TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **21.4%**

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A 23-year-old man presents with 2 days of chest pain to the emergency department. The pain is moderate and made worse if he breathes in. He is usually well but did have a few days off work for 'flu' last week.

On examination, he is sitting forward and groans in pain when asked to lie flat. His heart sounds are normal and his chest is clear. He is tender in his trapezius and scapular muscles but able to flex his neck .

Na^+	135 mmol/l
K^+	4.9 mmol/l
Urea	4.2 mmol/l
Creatinine	86 µmol/l

Hb	130 g/l
Platelets	354 * 10^9/l
WBC	4.5 * 10^9/l
Lymphocytes	0.1* 10^9/l

Chest X-ray	clear lung fields, normal heart contour

What ECG finding would be most specific in the likely diagnosis?

○ S1Q3T3

○ ST elevation

○ Sinus arrhythmia

○ Sinus tachycardia

○ PR depression

Submit answer

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Lymphocytes	$0.1* 10^9$/l

Chest X-ray	clear lung fields, normal heart contour

What ECG finding would be most specific in the likely diagnosis?

S1Q3T3	4%
ST elevation	10%
Sinus arrhythmia	1%
Sinus tachycardia	3%
PR depression	83%

The most specific ECG finding in acute pericarditis is PR depression

Important for me Less important

The chest pain that is worse on lying flat in a person with a recent viral illness is due to pericarditis. ST elevation and PR depression are seen with the latter being the most specific. Sinus tachycardia and S1Q3T3 pattern are more commonly seen in pulmonary embolism. Sinus arrhythmia is a normal phenomenon in which the heart rate varies with the respiratory cycle.

👍 👎 Discuss (1) Improve

Next question >

Acute pericarditis ★

Acute pericarditis is a condition referring to inflammation of the pericardial sac, lasting for less than 4-6 weeks.

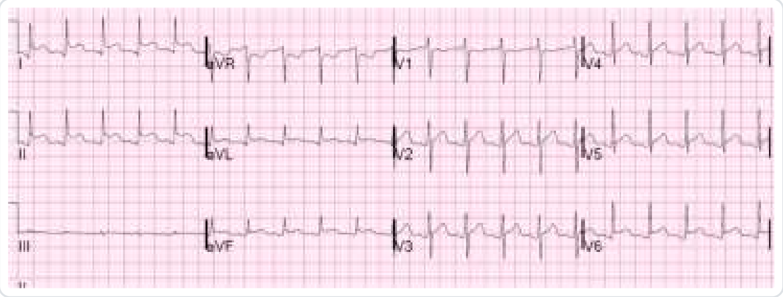
Aetiology
- viral infections (Coxsackie)
- tuberculosis
- uraemia
- post-myocardial infarction
  - early (1-3 days): fibrinous pericarditis
  - late (weeks to months): autoimmune pericarditis (Dressler's syndrome)
- radiotherapy
- connective tissue disease
  - systemic lupus erythematosus
  - rheumatoid arthritis
- hypothyroidism
- malignancy
  - lung cancer
  - breast cancer
- trauma

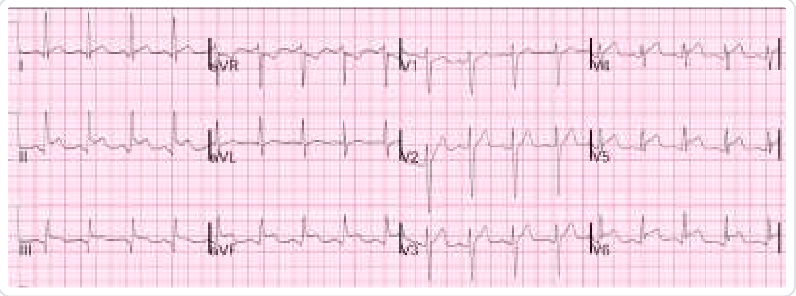
Features
- chest pain: may be pleuritic. Is often relieved by sitting forwards
- other symptoms include a non-productive cough, dyspnoea and flu-like symptoms

- pericardial rub

Investigations

- ECG changes
  - the changes in pericarditis are often global/widespread, as opposed to the 'territories' seen in ischaemic events
  - 'saddle-shaped' ST elevation
  - PR depression: most specific ECG marker for pericarditis
- all patients with suspected acute pericarditis should have transthoracic echocardiography
- bloods
  - inflammatory markers
  - troponin: around 30% of patients may have an elevated troponin - this indicates possible myopericarditis





Management

- the majority of patients can be managed as outpatients
  - patients who have high-risk features such as fever > 38°C or elevated troponin should be managed as an inpatient
- treat any underlying cause
  - most patients however will have pericarditis secondary to viral infection, meaning no specific treatment is indicated
- strenuous physical activity should be avoided until symptom resolution and normalisation of inflammatory markers
- a combination of NSAIDs and colchicine is now generally used for first-line for patients with acute idiopathic or viral pericarditis
  - until symptom resolution and normalisation of inflammatory markers (usually 1-2 weeks) followed by tapering of dose

recommended over

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane 36 15

Pericarditis review

Suggest link

Report broken link

Media

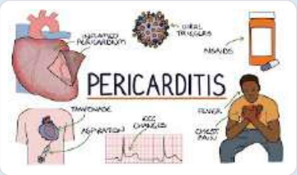
PERICARDITIS & PERICARDIAL EFFUSIONS
Osmosis.org

Pericarditis and pericardial effusions

Osmosis - YouTube 2 0

Pericardial rub - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 0


PERICARDITIS

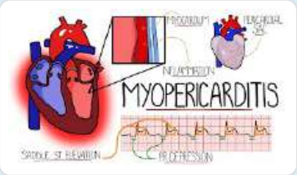
Pericarditis

Zero To Finals - YouTube 👍 0 👎 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 👍 2 👎 1


MYOPERICARDITIS

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

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117 ✗

118 ✗

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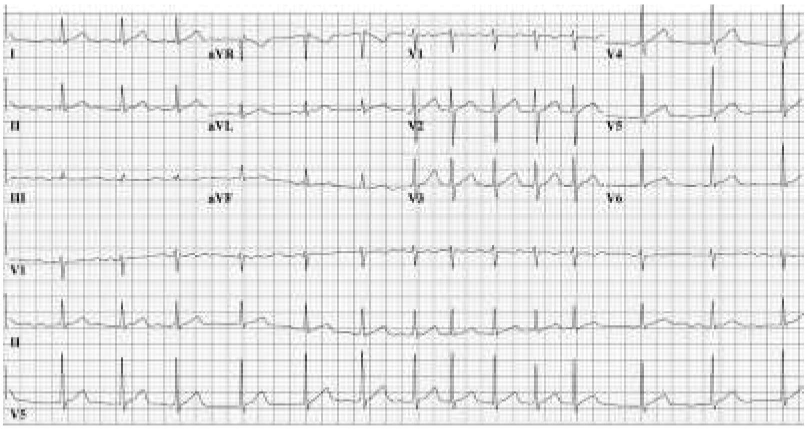
A 68-year-old man presents to hospital with left-sided upper and lower limb weakness that was present on waking that morning. The symptoms continue to persist in hospital. On examination, he has homonymous hemianopia. He has no significant medical history and is not on any regular medications.

A CT head is performed which is unremarkable.

An ECG is performed, which is shown below.



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How would you manage this patient's anti-thrombotic medication?

○ Apixaban for life

○ Aspirin 300mg once daily for 14 days, then apixaban for life

○ Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life

○ Clopidogrel 75mg once daily for life

○ Thrombolysis with alteplase, then apixaban for life

Submit answer

Reference ranges

Question 119 of 129 ✗

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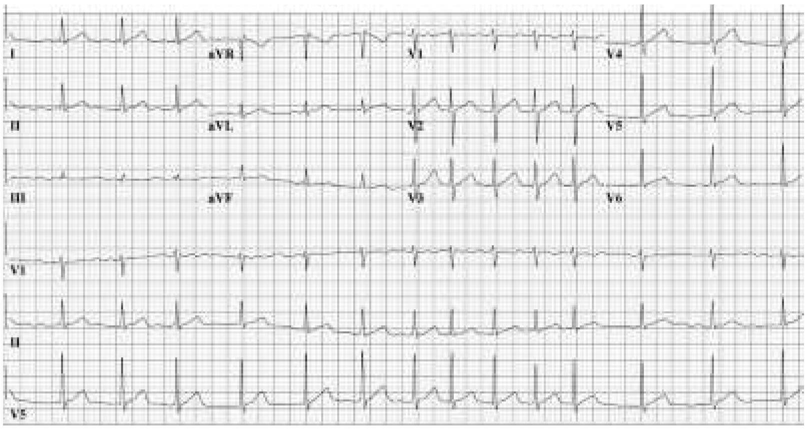
A CT head is performed which is unremarkable.

An ECG is performed, which is shown below.



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How would you manage this patient's anti-thrombotic medication?

Apixaban for life	12%
Aspirin 300mg once daily for 14 days, then apixaban for life	61%
Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life	12%
Clopidogrel 75mg once daily for life	4%
Thrombolysis with alteplase, then apixaban for life	12%

The ECG shows atrial fibrillation, as evidenced by the irregularly irregular rhythm and the lack of p-waves.

Aspirin 300mg once daily for 14 days, then apixaban for life is the correct answer. This patient has symptoms in keeping with a partial anterior circulation stroke. The CT head excludes an intracranial haemorrhage. Note, a normal scan is not sufficient to exclude a stroke, and stroke diagnosis should be based on clinical signs. The ECG shows atrial fibrillation which is the likely cause of the stroke. His CHA_2DS_2-VASc score is 3 due to age (+1) and stroke (+2). As such, he should receive two weeks of single anti-platelet treatment (aspirin 300mg once daily) as with all ischaemic strokes, followed by lifelong anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban, to prevent further strokes secondary to atrial fibrillation. Initiation of apixaban is delayed by two weeks post-stroke to prevent haemorrhagic transformation.

Apixaban for life is the incorrect answer. Therapeutic anticoagulation with a DOAC should not be initiated until after two weeks to avoid hemorrhagic transformation of an ischaemic stroke.

Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life is the incorrect answer. This would be the correct answer if this patient did not have atrial fibrillation. Clopidogrel is insufficiently anti-thrombotic to prevent strokes secondary to atrial fibrillation and therefore a lifelong DOAC is required.

Clopidogrel 75mg once daily for life is the incorrect answer. A higher dose of antiplatelet is required to initially treat an ischaemic stroke (aspirin 300mg) and a DOAC is required long-term to reduce the atrial fibrillation-associated stroke risk.

Thrombolysis with alteplase, then apixaban for life is the incorrect answer. This patient does not meet the criteria for thrombolysis as he awoke with the symptoms - thrombolysis requires symptom onset within the past four hours.

👍 👎 Discuss (6) Improve

Next question >

Atrial fibrillation: rate control and maintenance of sinus rhythm ★

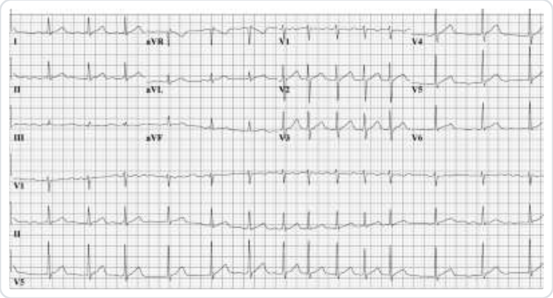
NICE updated its guidelines on the management of atrial fibrillation (AF) in 2021. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2012 guidelines

Patients presenting acutely with AF

If a patient has signs of haemodynamic instability (e.g. hypotension, heart failure) they should be electrically cardioverted, as per the peri-arrest tachycardia guidelines.

For haemodynamically stable patients, the management depends on how acute the AF is:

- < 48 hours: rate or rhythm control
- ≥ 48 hours or uncertain (e.g. patient not sure when symptoms started): rate control
  - if considered for long-term rhythm control, delay cardioversion until they have been maintained on therapeutic anticoagulation for a minimum of 3 weeks



Rate control

Rate control should be offered as the first-line treatment strategy for atrial fibrillation except in people:

- whose atrial fibrillation has a reversible cause
- who have heart failure thought to be primarily caused by atrial fibrillation
- with new-onset atrial fibrillation (< 48 hours)
- with atrial flutter whose condition is considered suitable for an ablation strategy to restore sinus rhythm
- for whom a rhythm-control strategy would be more suitable based on clinical judgement

Medications

Agents used to control rate in patients with atrial fibrillation

- beta-blockers
    - a common contraindication for beta-blockers is asthma
- calcium channel blockers
- digoxin

+ not considered first-line anymore as they are less effective at controlling the heart rate during exercise

- should only be considered if he person does no or very little physical exercise or other rate-limiting drug options are ruled out because of comorbidities
- may have a role if there is coexistent heart failure

Rhythm control

Agents used to maintain sinus rhythm in patients with a history of atrial fibrillation

- beta-blockers
- dronedarone: second-line in patients following cardioversion
- amiodarone: particularly if coexisting heart failure

Catheter ablation

NICE recommends the use of catheter ablation for those with AF who have not responded to or wish to avoid, antiarrhythmic medication.

Technical aspects

- the aim is to ablate the faulty electrical pathways that are resulting in atrial fibrillation. This is typically due to aberrant electrical activity between the pulmonary veins and left atrium
- the procedure is performed percutaneously, typically via the groin
- both radiofrequency (uses heat generated from medium frequency alternating current) and cryotherapy can be used to ablate the tissue

Anticoagulation

- should be used 4 weeks before and during the procedure

- it should be remember that catheter ablation controls the rhythm but does not reduce the stroke risk, even if patients remain in sinus rhythm. Therefore, patients still require anticoagulation as per their CHA$_2$DS$_2$-VASc score
  - if score = 0: 2 months anticoagulation recommended
  - if score > 1: longterm anticoagulation recommended

Outcome

- notable complications include
  - cardiac tamponade
  - stroke
  - pulmonary vein stenosis
- success rate
  - around 50% of patients experience an early recurrence (within 3 months) of AF that often resolves spontaneously
  - longer term, after 3 years, around 55% of patients who've had a single procedure remain in sinus rhythm. Of patients who've undergone multiple procedures around 80% are in sinus rhythm

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

European Society of Cardiology 👍 1 👎 1

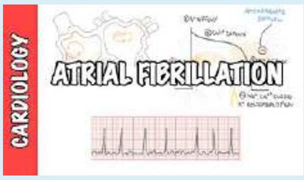
2020 Atrial fibrillation guidelines

NICE 👍 1 👎 2

2021 Atrial fibrillation guidelines

Suggest link Report broken link

Media


CARDIOLOGY
ATRIAL FIBRILLATION

Atrial Fibrillation Overview - ECG, types, pathophysiology, treatment, complications

Armando Hasudungan - YouTube 👍 0 👎 0

Report broken media

Score: **21%**

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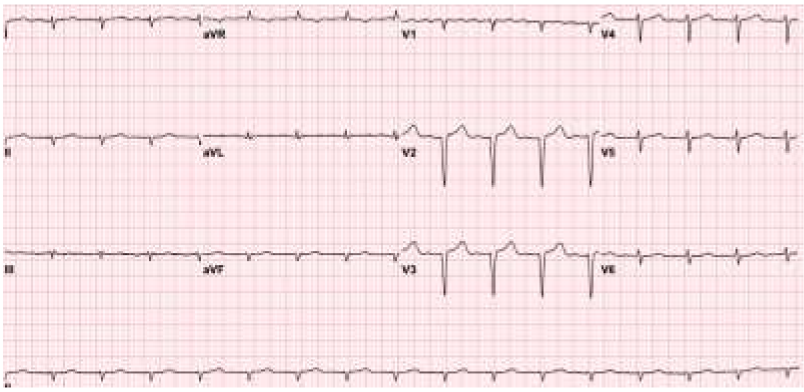
An 81-year-old man is referred to the cardiology clinic due to worsening heart failure. Over the past 12 months he has become increasingly short-of-breath, particularly upon exertion or when lying flat. He has also suffered from ankle swelling which his GP has treated with large doses of furosemide. Around 6 months ago a clinical diagnosis of heart failure was made and he is already taking bisoprolol, ramipril and atorvastatin. He was diagnosed with multiple myeloma around 3 years and has been treated with melphalan to date.

On examination his pulse is 72/min and blood pressure 98/62 mmHg. The JVP is elevated at 6cm above the angle of Louis. There are crackles in both bases and pitting oedema to his knees.

An ECG accompanies the referral:



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What is the most likely diagnosis?

○ Constrictive pericarditis ×

○ Infective endocarditis ×

○ Chronic pulmonary embolism ×

○ Cardiac amyloidosis ×

○ Hypercalcaemia-induced heart failure ×

Submit answer

Reference ranges

Score: **21%**

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Question 120 of 129 ×

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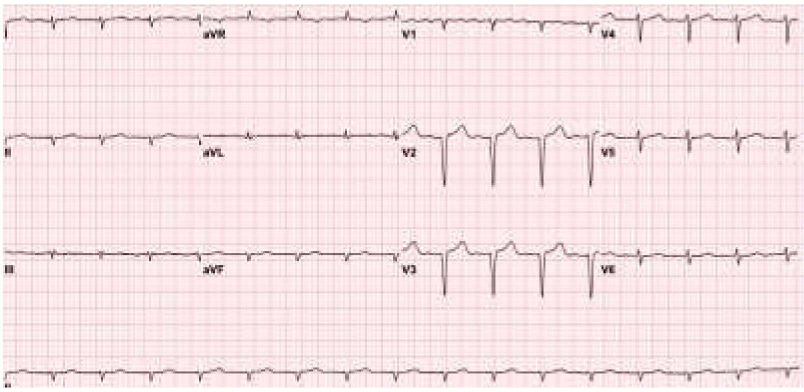

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What is the most likely diagnosis?

- Constrictive pericarditis 28%
- Infective endocarditis 0%
- Chronic pulmonary embolism 2%
- Cardiac amyloidosis 65%
- Hypercalcaemia-induced heart failure 5%

This patient is likely to have AL amyloidosis secondary to his myeloma. This has resulted in cardiac amyloidosis as evidenced by the low voltage QRS complexes asssociated with poor R wave progression in the chest leads (a pseudoinfarction pattern).

Discuss (10) | Improve

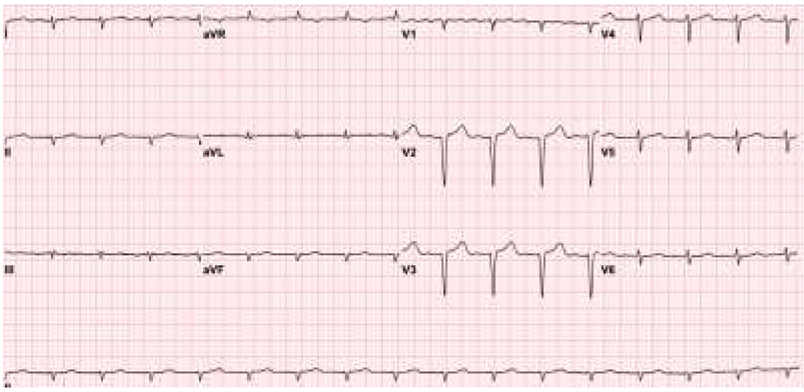
Next question >

Amyloidosis: cardiac

Cardiac amyloidosis, also known as amyloid cardiomyopathy, is a rare disorder characterized by the extracellular deposition of insoluble amyloid fibrils in the myocardium, leading to progressive heart failure and conduction abnormalities. Cardiac amyloidosis can occur in isolation or as part of systemic amyloidosis, which affects multiple organs.

Investigations

- ECG: typically shows low-voltage complexes with poor R wave progression in the chest leads (a pseudoinfarction pattern).
- echocardiogram: increased myocardial echogenicity (granular sparkling or 'global speckled' appearance)



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+ Q 123

Next question >

Textbooks

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Links

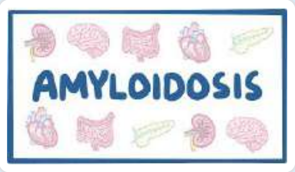
Journal of the American Heart Association 👍 6 👎 0

[Updates in Cardiac Amyloidosis: A Review]

Suggest link

Report broken link

Media



Amyloidosis

Osmosis - YouTube 👍 17 👎 2

Report broken media

Score: **20.8%**

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A 52-year-old lady was admitted with sudden onset central chest pain and breathlessness. Her past medical history included hypertension, atrial fibrillation and she was a smoker of 20 cigarettes a day. She lived alone after having recently split up with her husband which had been a difficult few weeks. Her friend had been staying with her occasionally during this difficult period.

She had a family history of ischaemic heart disease. She had recently complained to her friend of a cold that was now passing. Medications included aspirin 75mg OD and bisoprolol 5mg OD. Her friend called 999 and she was admitted directly to the angiogram suite as the paramedics noticed that her ECG showed ST elevation in the anterior chest leads.

Her angiogram showed mild coronary atherosclerosis but an akinetic left ventricle. Her troponin T was significantly elevated at 7800ng/L (normal < 14).

What is the most likely diagnosis?

○ Pericarditis ×

○ Myocardial infarction ×

○ Embolic event ×

○ Takotsubo cardiomyopathy ×

○ Pneumothorax ×

Submit answer

Reference ranges

Score: **20.8%**

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Her angiogram showed mild coronary atherosclerosis but an akinetic left ventricle. Her troponin T was significantly elevated at 7800ng/L (normal < 14).

What is the most likely diagnosis?

Pericarditis	7%
Myocardial infarction	13%
Embolic event	2%
Takotsubo cardiomyopathy	78%
Pneumothorax	0%

Takotsubo cardiomyopathy (also known as broken heart syndrome, apical ballooning syndrome, acute stress cardiomyopathy) is non-ischaemic cardiomyopathy where there is a temporary weakening of the heart muscle. This is often triggered by emotional stress or physical stress. Takotsubo cardiomyopathy is more commonly seen in post-menopausal women Patients most commonly present with chest pain and shortness of breath. Sudden onset congestive cardiac failure may also occur.

On initial presentation diagnosis of Takotsubo cardiomyopathy may be difficult and patients are often treated as an acute myocardial infarction. ECGs classically mimics ST-segment elevation myocardial infarction. Patients often proceed acutely to an angiogram which shows the absence of significant heart disease. Cardiac enzymes are significantly raised.

An echocardiogram may demonstrate pathognomonic wall motion abnormalities, in which the base of the left ventricle is contracting normally or is hyperkinetic while the remainder of the left ventricle is akinetic or dyskinetic. This coupled with the lack of significant coronary artery disease that would explain the wall motion abnormalities allows the diagnosis to be made.

Treatment of Takotsubo cardiomyopathy is generally supportive. Patients may be hypotensive, however, treatment with inotropes often worsens the disease. Patients may require intra-aortic balloon pump, fluids, beta blockers or calcium channel blockers. Aspirin is also commonly used in the management. Once over the initial event outcomes are favourable. Even when ventricular systolic function is heavily compromised at presentation, it typically improves within the first few days and normalises within the first few months

👍 👎 Discuss (6) Improve

Next question >

Takotsubo cardiomyopathy ★

Takotsubo cardiomyopathy is a type of non-ischaemic cardiomyopathy associated with a transient, apical ballooning of the myocardium. It may be triggered by stress.

Pathophysiology

- Takotsubo is a Japanese word that describes an octopus trap
- the apical ballooning appearance occurs due to severe hypokinesis of the mid and apical segments with preservation of activity of the basal segments. In simple terms, the bottom of the heart (the apex) does not contract and therefore appears to balloon out. However,

the area closer to the top (the base) continues to contract (creating the neck of the octopus trap)

Features
- chest pain
- features of heart failure
- ECG: ST-elevation
- normal coronary angiogram

Treatment is supportive.

Prognosis
- the majority of patients improve with supportive treatment

123

Next question

Textbooks

High-yield textbook

Extended textbook

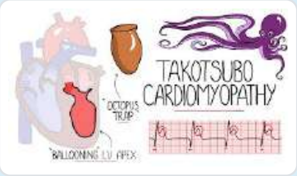
Media

CARDIOLOGY
TAKOTSUBO CARDIOMYOPATHY

Takotsubo cardiomyopathy

Armando Hasudungan - YouTube

👍 4 👎 0


TAKOTSUBO CARDIOMYOPATHY
OCTOPUS TRAP
BALLOONING LV APEX

Takotsubo cardiomyopathy

Rhesus Medicine - YouTube 👍 0 👎 0

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Score: **20.7%**

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115	✗
116	✗
117	✗
118	✗
119	✗
120	✗
121	✗

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A 71-year-old gentleman is reviewed in hospital prior to discharge after having received treatment for an exacerbation of COPD. His systolic blood pressure has been recorded as measuring between 138mmHg to 156mmHg. He has a background of ischaemic heart disease, gout, gallstones and a fractured neck of femur which was repaired with a dynamic hip screw. He is found to have ACR of 31mg/mmol. He normally takes allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

What is the most appropriate pharmacological management for his hypertension?

- ○ ACE-inhibitor
- ○ Calcium channel blocker
- ○ Thiazide-like diuretic
- ○ Beta-blocker
- ○ Alpha-blocker

Submit answer

Reference ranges

Score: **20.7%**

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A 71-year-old gentleman is reviewed in hospital prior to discharge after having received treatment for an exacerbation of COPD. His systolic blood pressure has been recorded as measuring between 138mmHg to 156mmHg. He has a background of ischaemic heart disease, gout, gallstones and a fractured neck of femur which was repaired with a dynamic hip screw. He is found to have ACR of 31mg/mmol. He normally takes allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

What is the most appropriate pharmacological management for his hypertension?

ACE-inhibitor	66%
Calcium channel blocker	31%
Thiazide-like diuretic	2%
Beta-blocker	1%
Alpha-blocker	0%

The correct answer is ACE-inhibitor. This is an elderly man with hypertension and proteinuria. A calcium channel blocker would have been appropriate if he did not have proteinuria, whilst a thiazide-like diuretic and alpha-blocker feature at later stages in the NICE guidelines. Beta-blockers can provoke bronchospasms in COPD.

Source:

Lewington, Andrew, and Suren Kanagasundaram. 'Renal Association Clinical Practice Guidelines on Acute Kidney Injury.' Nephron Clinical Practice 118.S1 (2011): C349-390

Discuss (8) Improve

Next question >

Hypertension: management

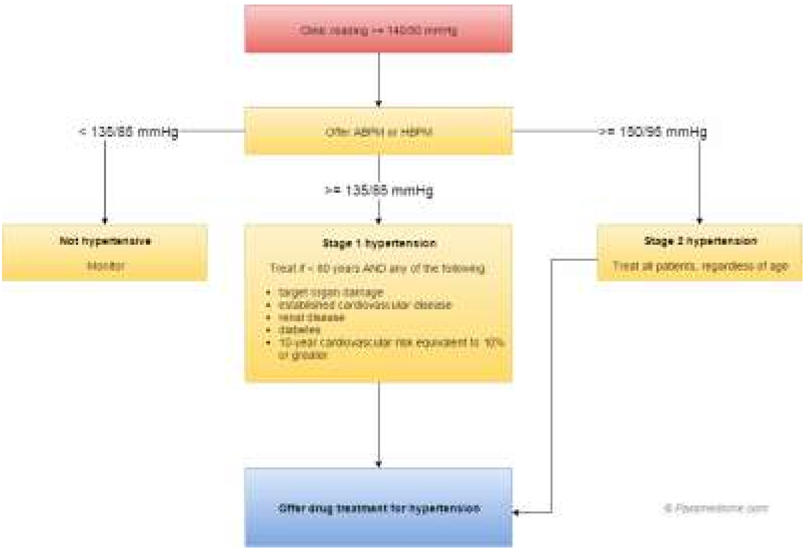
NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
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Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

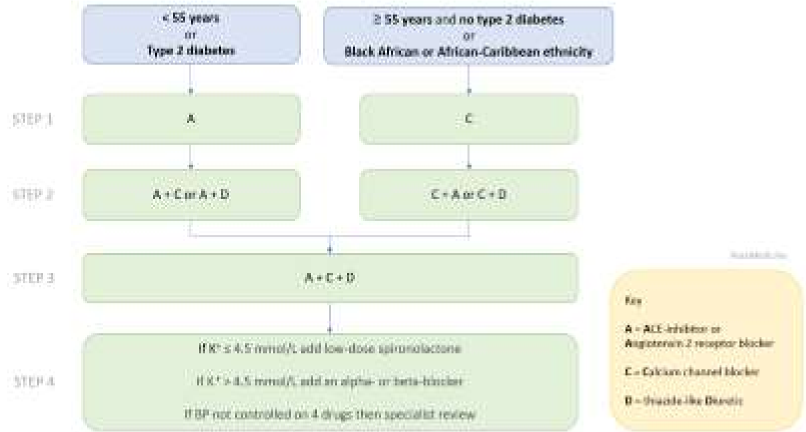
ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)

- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment

- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
  - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor

- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

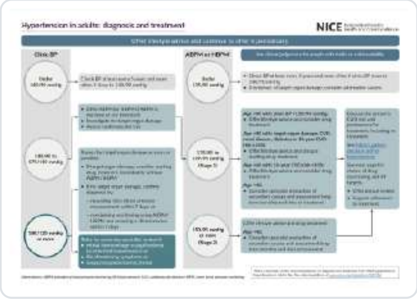
- add a third drug to make, i.e.:
  - if already taking an **(A + C)** then add a **D**
  - if already **(A + D)** then add a **C**
- **(A + C + D)**

Step 4 treatment

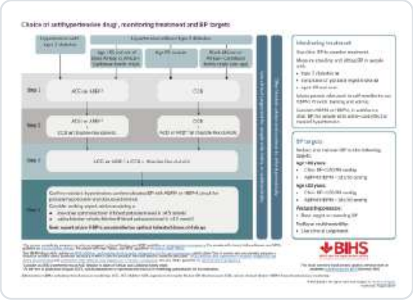
- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
  - confirm elevated clinic BP with ABPM or HBPM
  - assess for postural hypotension.
  - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*



Hypertension in adults: diagnosis and treatment



Choice of antihypertensive drug, monitoring treatment and BP targets

Blood pressure targets

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE

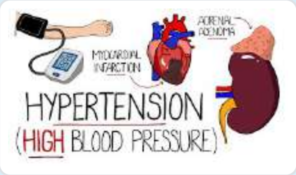
👍 162 👎 162

2019 Hypertension guidelines

Suggest link

Report broken link

Media


MYOCARDIAL INFARCTION
ADRENAL ADENOMA
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

Report broken media

Score: **21.3%**

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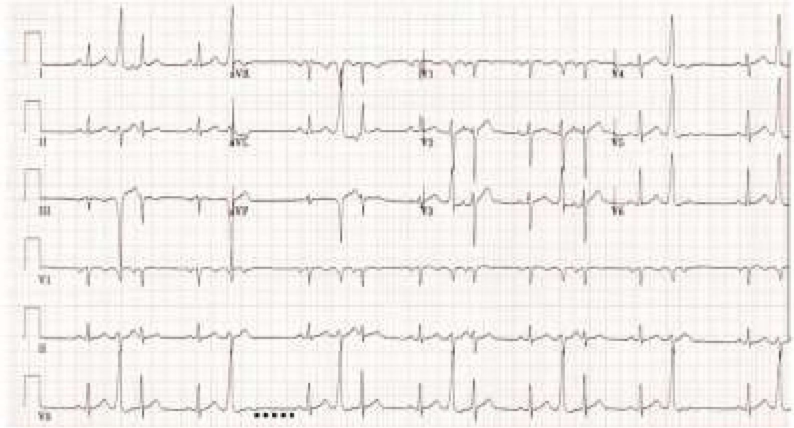
Question 123 of 129

A 58-year-old woman presents to the emergency department complaining of palpitations for half an hour. She has been diagnosed with diabetes, heart failure and depression and is on metformin, dapagliflozin, losartan, digoxin and sertraline. She reports experiencing abdominal cramps and loose stools over the last two days, which began following dinner at a restaurant. Her heart rate is 103 beats per minute, her BP is 100/70 mmHg, and respiratory rate is 20 breaths/min. An ECG was taken:



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What is the most likely cause of the findings on the ECG?

○ Anxiety

○ Dapagliflozin

○ Digoxin

○ Metformin

○ Sertraline

Submit answer

Reference ranges

Score: **21.3%**

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Question 123 of 129 ×

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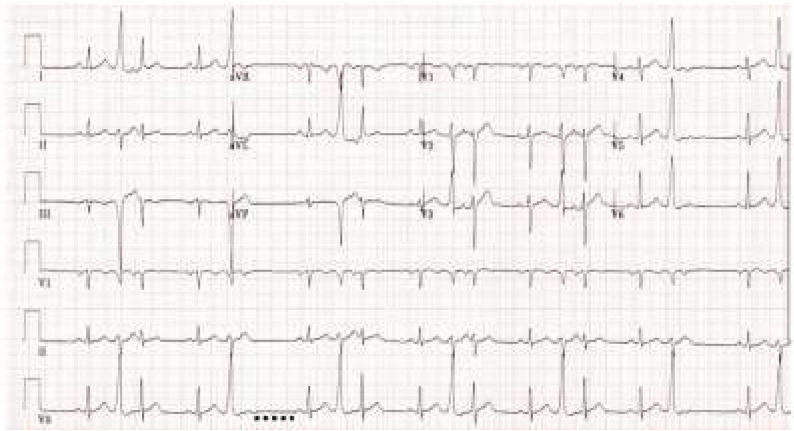


© Image used on license from Dr Smith, University of Minnesota

What is the most likely cause of the findings on the ECG?

- Anxiety 5%
- Dapagliflozin 3%
- Digoxin 62%
- Metformin 3%
- Sertraline 26%

The correct answer is **digoxin**. The electrocardiogram (ECG) presented demonstrates premature ventricular complexes (PVCs), which are characterised by abnormally large QRS complexes that disrupt the underlying sinus rhythm. A typical PVC is identifiable on an ECG as a broad QRS complex, exceeding 120 milliseconds in duration, with an abnormal morphology. It presents prematurely and is associated with discordant ST segment and T wave changes, followed by a compensatory pause prior to the next normal sinus impulse. Digoxin toxicity is recognised for inducing a spectrum of arrhythmias that may be life-threatening. Arrhythmias associated with digoxin toxicity include paroxysmal atrial tachycardia, atrioventricular (AV) blocks, junctional rhythms, and ventricular arrhythmias such as PVCs and ventricular tachycardia. Notably, PVCs are often the initial indicator of digoxin toxicity and represent the most frequent arrhythmia linked to elevated serum digoxin levels. In this case, it is probable that hypokalaemia induced by diarrhoea has precipitated the digoxin toxicity.

Anxiety can lead to the emergence of PVCs; however, it is not deemed the primary cause in this clinical scenario. Anxiety-related PVCs are thought to originate from sympathetic nervous system overactivity. Nonetheless, given the circumstances described above, digoxin toxicity remains a more plausible explanation for this patient's presentation.

Metformin is not associated with arrhythmias when used within therapeutic dosages. Metformin toxicity might arise in contexts such as renal impairment or intentional overdose and could result in severe lactic acidosis that may precipitate arrhythmias. Despite this possibility, hypokalaemia secondary to diarrhoea resulting in digoxin toxicity appears to be a more convincing cause for the observed PVCs.

Sertraline, an selective serotonin reuptake inhibitor (SSRI), is utilised primarily in managing depression and does not align with this clinical picture as a causative agent for arrhythmia. Although sertraline is among the preferred antidepressants following myocardial infarction (MI), there exists no direct association between sertraline use and arrhythmic events. It should be noted that certain psychiatric medications such as clozapine, chlorpromazine and thioridazine have been implicated in arrhythmic side effects.

Dapagliflozin stands incorrect for this question. As an SGLT-2 inhibitor prescribed for heart failure and diabetes management, dapagliflozin has been reported to reduce incidents of significant ventricular arrhythmias including cardiac arrest or sudden death; thus it would be improbable for dapagliflozin to account for the clinical findings described here.

👍 👎 Discuss (2) Improve

Next question >

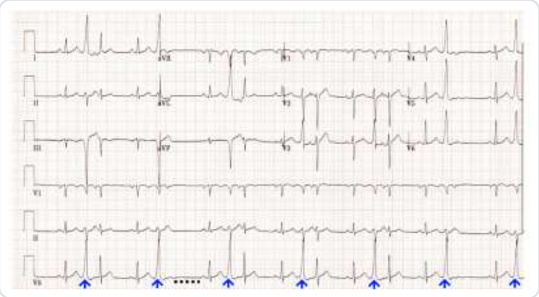
Premature ventricular complexes

Premature ventricular complexes (PVCs; also known as premature ventricular beats and ventricular extrasystoles) refer to beats originating in the ventricular myocardium. They are common are occur in a wide population group, including those with and without underlying cardiac disease.

Aetiology
- idiopathic: the vast majority
- hypertension
- myocardial infarction

Features
- the vast majority are asymptomatic
- some patients experience palpitations, typically the sensation of a 'skipped beat'
- the pulse will feel irregular during regular PVCs
- ECG
    - as the beats originate in the ventricle they form a wide QRS

Management
- asymptomatic patients with no evidence of underlying cardiac disease do not require any further management



123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **21.1%**

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117	✗
118	✗
119	✗
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121	✗
122	✓
123	✗

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Question 124 of 129

An 86-year-old female presents to the diabetic ulcer clinic for regular foot care when regular observations note her heart rate to be 42 beats per minute. Her blood pressure is 142/55 mmHg and she reports no recent episodes of syncope. A 12 lead ECG demonstrates Mobitz type 1 rhythm at 42 beats/ minute. A 24 hours tape performed as an outpatient demonstrates bradycardia with up to 2.7 second pauses. Her past medical history includes type 2 diabetes mellitus, hypertension and angina. Her medications include ramipril, furosemide, verapamil, metformin and GTN on an as-required basis. During this second encounter, her heart rate is 41 beats/min and her blood pressure is 115/ 57 mmHg. She informs you that she is guided by you in terms of the most appropriate treatment. What do you advise?

- ○ Temporary pacing wire ×
- ○ Permanent pacemaker insertion ×
- ○ Stop verapamil ×
- ○ Regular digoxin ×
- ○ Stop furosemide ×

Submit answer

Reference ranges

Score: **21.1%**

1 ✗
2 ✓
3 ✗

4	✗
5	✓
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An 86-year-old female presents to the diabetic ulcer clinic for regular foot care when regular observations note her heart rate to be 42 beats per minute. Her blood pressure is 142/55 mmHg and she reports no recent episodes of syncope. A 12 lead ECG demonstrates Mobitz type 1 rhythm at 42 beats/ minute. A 24 hours tape performed as an outpatient demonstrates bradycardia with up to 2.7 second pauses. Her past medical history includes type 2 diabetes mellitus, hypertension and angina. Her medications include ramipril, furosemide, verapamil, metformin and GTN on an as-required basis. During this second encounter, her heart rate is 41 beats/min and her blood pressure is 115/ 57 mmHg. She informs you that she is guided by you in terms of the most appropriate treatment. What do you advise?

Temporary pacing wire	5%
Permanent pacemaker insertion	16%
Stop verapamil	77%
Regular digoxin	1%
Stop furosemide	1%

The question focuses on the management of bradycardia and indications for pacemaker insertion. The patient is asymptomatic and sustains a reasonable blood pressure, there is no indication for a temporary pacing wire. Digoxin would only further the atrioventricular node block. Stopping furosemide is unlikely to affect the heart rate. Indications for pacemaker insertion is dependent on whether the patient has recently suffered a myocardial infarction. For patients post-MI, complete heart block, Mobitz type II, trifascicular block, symptomatic bradycardia or bilateral bundle branch block. For a patient not post-MI, symptomatic second and third-degree heart block or with haemodynamic compromise or VT secondary to bradycardia are indications. In this case, the patient is not post-MI and asymptomatic with no haemodynamic compromise. There are no indications for a pacemaker, stopping verapamil, an AV node blocker should be sufficient.

1. Dual-chamber pacemakers for the treatment of symptomatic bradycardia, NICE Technology Appraisal. February 2005

👍 👎 Discuss (6) Improve

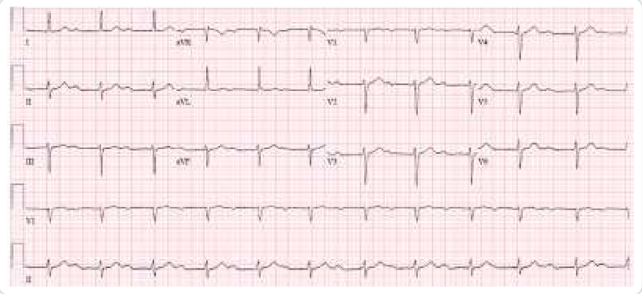
Next question >

Atrioventricular block ★

In atrioventricular (AV) block, or heart block, there is impaired electrical conduction between the atria and ventricles. There are three types:

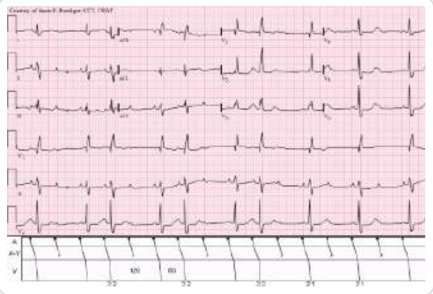
First-degree heart block

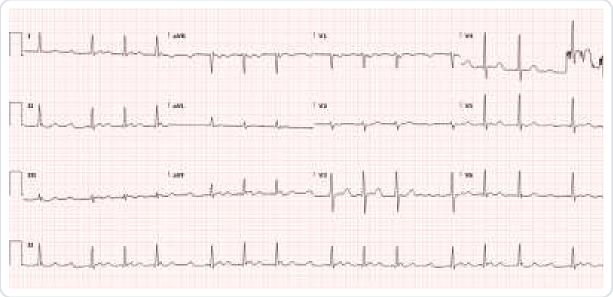
- PR interval > 0.2 seconds
- asymptomatic first-degree heart block is relatively common and does not need treatment



Second-degree heart block

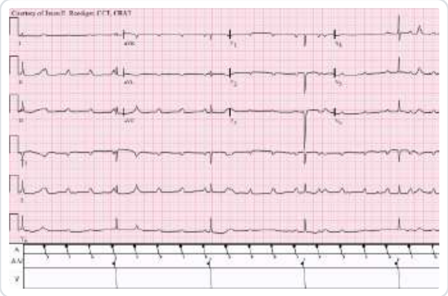
- type 1 (Mobitz I, Wenckebach): progressive prolongation of the PR interval until a dropped beat occurs
- type 2 (Mobitz II): PR interval is constant but the P wave is often not followed by a QRS complex

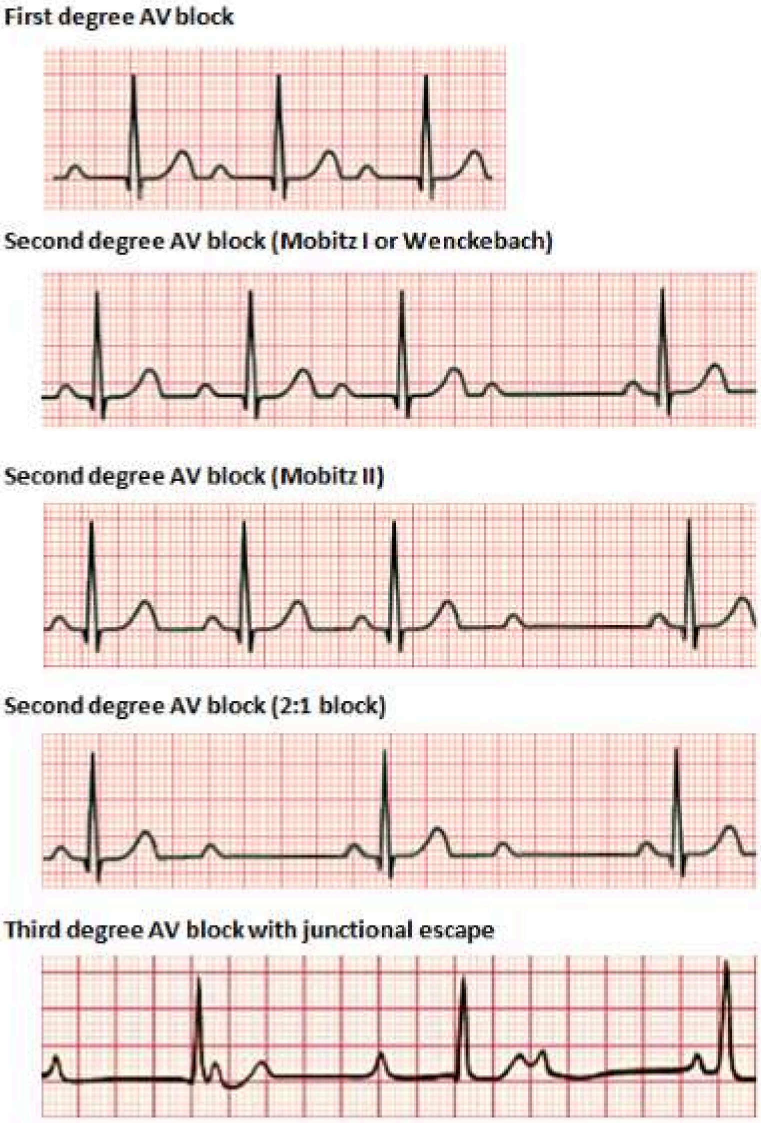




Third-degree (complete) heart block

- there is no association between the P waves and QRS complexes





First degree AV block

Second degree AV block (Mobitz I or Wenckebach)

Second degree AV block (Mobitz II)

Second degree AV block (2:1 block)

Third degree AV block with junctional escape

ECG showing types of heart block

123

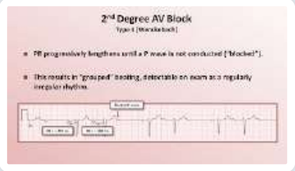
Next question

Textbooks

High-yield textbook

Extended textbook

Media


2nd Degree AV Block

AV block

Strong Medicine - YouTube

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Atrioventricular block

Osmosis - YouTube

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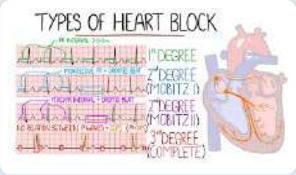
TYPES OF HEART BLOCK
1st DEGREE
2nd DEGREE (MOBITZ I)
2nd DEGREE (MOBITZ II)
3rd DEGREE (COMPLETE)

Heart Blocks Made Easy - 1st, 2nd (Mobitz 1/Wenckebach & Mobitz 2), 3rd (Complete)

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **21%**

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A 50-year-old gentleman presented feeling dizzy and generally unwell for the last 3 days. He denied any chest pain, cough or fever but complained of increased fatigue and breathlessness. His past medical history included ischaemic heart disease, heart transplant, hypertension and diabetes. He takes the following medications: ramipril 5mg od, metformin 500mg bd, simvastatin 40mg od, amlodipine 5mg od and various immunosuppressive agents.

On examination his heart sounds were normal and chest was clear. There was only mild leg oedema. His vital signs show heart rate = 35 beats per minute, blood pressure = 90/40, SaO2 = 95% on air, T= 36.5°C and respiratory rate = 20 breaths per minute. His ECG shows sinus bradycardia and CXR is clear. The following blood tests were obtained:

Hb	10.5 g/dl
Platelets	190 * 10^9/l
WBC	10.4 * 10^9/l

Na^+	135 mmol/l
K^+	3.6 mmol/l
Urea	8 mmol/l
Creatinine	100 µmol/l
CRP	15 mg/l

What is the next best management plan?

- ○ Glucagon intravenous ×
- ○ Atropine intravenous ×
- ○ Theophyline intravenous ×
- ○ Adrenaline 0.5mg intravenous ×

○ Adrenaline 0.5mg intramuscular ×

Submit answer

Reference ranges ⌄

Score: **21%**

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Urea	8 mmol/l
Creatinine	100 µmol/l
CRP	15 mg/l

What is the next best management plan?

Glucagon intravenous	10%
Atropine intravenous	72%
Theophylline intravenous	12%
Adrenaline 0.5mg intravenous	4%

Adrenaline 0.5mg intramuscular 2%

This question aims to raise awareness about the treatment of bradycardia and some potential pitfalls.

According to ALS protocol any bradycardia has to be assessed according to the ABCDE principle. In the presence of shock, syncope, myocardial ischaemia or heart failure further treatment is needed as soon as possible. Atropine is usually the first line treatment for bradycardia and if that fails then consider transcutaneous or transvenous pacing. However, in patients with heart transplant atropine is contraindicated. The hearts of these patients are denervated and do not respond to vagal blockade by atropine, which might precipitate paradoxical sinus arrest of high-grade block. In these cases, theophyline intravenously is indicated.

Glucagon is indicated if the bradycardia is due to beta-blockers or calcium channel blockers. Adrenaline is a potential option but the dose mentioned is wrong and certainly use of intravenous adrenaline outside the cardiac arrest scenario requires expert help (intramuscular in the anaphylaxis scenario.

👍 👎 Discuss (16) Improve

Next question >

Peri-arrest rhythms: bradycardia ★

The 2021 Resuscitation Council (UK) guidelines emphasise that the management of bradycardia depends on:

- 1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
- 2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired

consciousness
- syncope
- myocardial ischaemia
- heart failure

Atropine (500mcg IV) is the first line treatment in this situation.

If there is an unsatisfactory response the following interventions may be used:

- atropine, up to a maximum of 3mg
- transcutaneous pacing
- isoprenaline/adrenaline infusion titrated to response

Specialist help should be sought for consideration of transvenous pacing if there is no response to the above measures.

Resuscitation Council UK

GUIDELINES 2021

Adult bradycardia

Potential risk of asystole

The following are risk factors for asystole. Even if there is a satisfactory response to atropine specialist help is indicated to consider the need for transvenous pacing:

- complete heart block with broad complex QRS
- recent asystole
- Mobitz type II AV block
- ventricular pause > 3 seconds

123

Next question >

Textbooks

High-yield textbook

Extended textbook

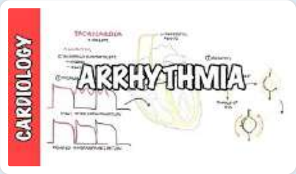
Links

Resuscitation Council UK 👍 22 👎 10

2021 Bradycardia algorithm

Suggest link Report broken link

Media



Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube 👍 2 👎 0

Report broken media

Score: **20.8%**

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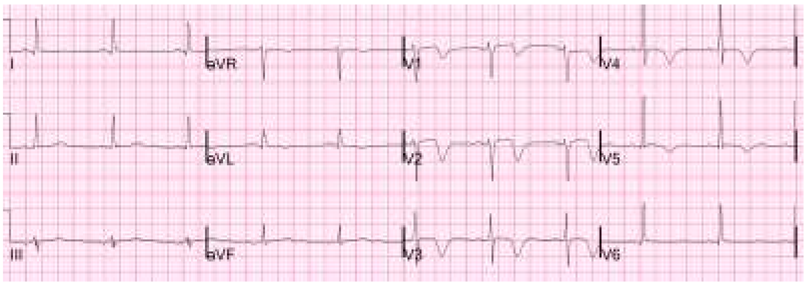
An ECG is taken (as shown below):



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What is the most likely diagnosis?

○ Brugada syndrome

○ Hypertrophic obstructive cardiomyopathy

○ Posterior myocardial infarction

○ Pulmonary embolus with right heart strain

○ Wellen's syndrome

Submit answer

Reference ranges

Score: **20.8%**

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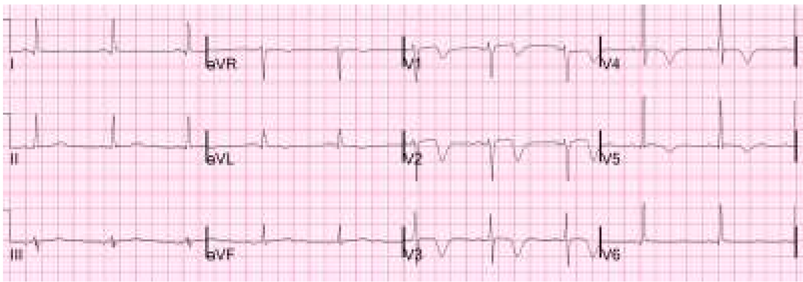


© Image used on license from Dr Smith, University of Minnesota

What is the most likely diagnosis?

Brugada syndrome	12%
Hypertrophic obstructive cardiomyopathy	6%
Posterior myocardial infarction	15%
Pulmonary embolus with right heart strain	5%
Wellen's syndrome	63%

This patient has **Wellen's syndrome**. Wellen's syndrome is the critical ischaemia of the left anterior descending artery. Patients typically have a history of chest pain and ECG findings include biphasic T waves in the anterior leads or deep symmetrical T wave inversion in leads I and aVL associated with 1mm ST elevation in the chest leads. These can be seen in this patient's ECG.

Brugada syndrome is incorrect. Brugada syndrome is an autosomal dominant inherited cardiovascular disease. It can be asymptomatic and lead to sudden cardiac death. ECG changes consistent with Brugada syndrome include convex ST-segment elevation > 2mm in at least 1 of V1-V3 that is followed by T wave inversion. A partial right bundle branch block may also be seen. Sometimes, a patient may have an appearance of a normal electrocardiogram (ECG). However, following the administration of flecainide, these ST-segment changes may appear.

Hypertrophic obstructive cardiomyopathy (HOCM) is incorrect. HOCM is an autosomal dominant inherited condition resulting in left ventricular hypertrophy with decreased cardiac compliance and ultimately decreased cardiac output. Most cases can be asymptomatic. However, symptoms include exertional dyspnoea or chest pain, syncope and sudden cardiac death. Given the family history of sudden cardiac death, HOCM is a reasonable differential. However, ECG features of HOCM include features of left ventricular hypertrophy (eg increased R wave height), deep Q waves and non-specific ST segment changes that are not seen in this patient's ECG.

Posterior myocardial infarction is incorrect. A posterior myocardial infarction causes ECG findings such as a dominant R wave in V2, and horizontal ST depression and broad R waves in V1-V3. Although the patient has cardiac-sounding chest pain, their ECG findings do not match those that we see here, making this answer incorrect.

Pulmonary embolus with right heart strain is incorrect. Classic findings of a pulmonary embolus include sinus tachycardia and the less common S1Q3T3 finding. However, in more significant disease, patients may exhibit features of **right heart strain** secondary to pulmonary hypertension. Right heart strain appears as ischaemic changes (including deep T wave inversion) in the right-sided leads of the heart. The history here is not suggestive of a pulmonary emboli and does not explain the biphasic T waves seen in V1 and V2.

👍 👎 Discuss (4) Improve

Next question >

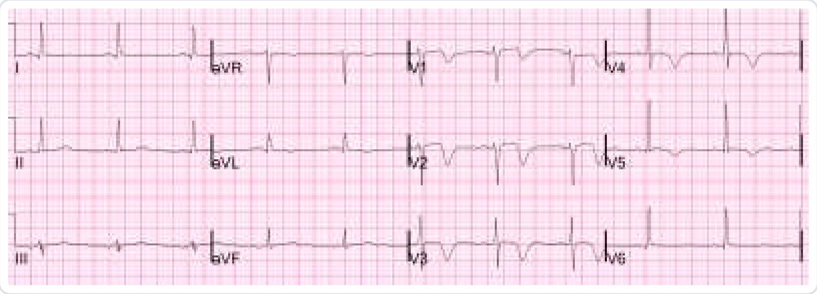
ECG: Wellen's syndrome ★

Wellen's syndrome is an ECG pattern that is typically caused by high-grade stenosis in the left anterior descending coronary artery.

The patient's pain may have resolved at the time of presentation and cardiac enzymes may be normal/minimally elevated.

ECG features

- biphasic or deep T wave inversion in V2-3
- minimal ST elevation
- no Q waves



+ Q 123

Next question ›

B *I*

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 6 1

Wellens Syndrome

Suggest link Report broken link

Score: **21.4%**

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Question 127 of 129

A 48-year-old man is brought in by ambulance to the Emergency Department after experiencing sudden onset crushing central chest pain radiating down his arm. His ECG showed an inferior STEMI and he undergoes successful PCI with 2 stents inserted into his right coronary artery. He is transferred to CCU and started on secondary prevention medication.

24 hours after his presentation he becomes bradycardic at 34bpm and his blood pressure falls to 80/47 mmHg. He denies chest pain but feels dizzy and light headed. His cardiac monitor shows 3rd-degree heart block. He is given atropine but his heart rate does not raise and his blood pressure remains low.

What is the most appropriate next step in his management?

- ○ Isoprenaline infusion ×
- ○ Temporary pacing wire ×
- ○ Permanent pacemaker ×
- ○ Repeat angiography ×
- ○ Adrenaline ×

Submit answer

Reference ranges

Score: **21.4%**

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Question 127 of 129

A 48-year-old man is brought in by ambulance to the Emergency Department after experiencing sudden onset crushing central chest pain radiating down his arm. His ECG showed an inferior STEMI and he undergoes successful PCI with 2 stents inserted into his right coronary artery. He is transferred to CCU and started on secondary prevention medication.

24 hours after his presentation he becomes bradycardic at 34bpm and his blood pressure falls to 80/47 mmHg. He denies chest pain but feels dizzy and light headed. His cardiac monitor shows 3rd-degree heart block. He is given atropine but his heart rate does not raise and his blood pressure remains low.

What is the most appropriate next step in his management?

Isoprenaline infusion	18%
Temporary pacing wire	69%
Permanent pacemaker	9%
Repeat angiography	2%
Adrenaline	2%

This gentleman has suffered an inferior STEMI. Complete heart block is a recognised complication of both inferior and anterior STEMI. Inferior STEMI CHB is usually more transient and they only require treatment if they become symptomatic. Anterior STEMI CHB is a more serious complication and they are more likely to need a permanent pacing system. In this case, as he has not responded to atropine then isoprenaline infusion is unlikely to work. There is no suggestion of repeat infarction so angiography is not indicated. He will require a temporary pacing wire to be placed.

Discuss (13) | Improve

Next question >

Peri-arrest rhythms: bradycardia

The 2021 Resuscitation Council (UK) guidelines emphasise that the management of bradycardia depends on:

- 1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
- 2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

- shock: hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness
- syncope
- myocardial ischaemia
- heart failure

Atropine (500mcg IV) is the first line treatment in this situation.

If there is an unsatisfactory response the following interventions may be used:

- atropine, up to a maximum of 3mg
- transcutaneous pacing
- isoprenaline/adrenaline infusion titrated to response

Specialist help should be sought for consideration of transvenous pacing if there is no response to the above measures.

Potential risk of asystole

The following are risk factors for asystole. Even if there is a satisfactory response to atropine specialist help is indicated to consider the need for transvenous pacing:

- complete heart block with broad complex QRS
- recent asystole
- Mobitz type II AV block
- ventricular pause > 3 seconds

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

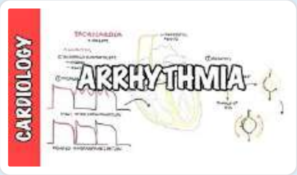
Links

Resuscitation Council UK 👍 22 👎 10

2021 Bradycardia algorithm

Suggest link Report broken link

Media


CARDIOLOGY
ARRHYTHMIA

Arrhythmia Overview - Mechanism of bradyarrhythmia and tachyarrhythmia

Armando Hasudungan - YouTube

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A 70-year-old woman with a history of type 2 diabetes mellitus and hypertension is reviewed in clinic. You can see from the records their is no evidence of diabetic retinopathy, chronic kidney disease or cardiovascular disease.

Her current medication is as follows:

- simvastatin 40mg on
- ramipril 10mg od
- amlodipine 5mg od
- metformin 1g bd

Recent blood results are shown below:

Na^+	142 mmol/l
K^+	4.4 mmol/l
Urea	7.2 mmol/l
Creatinine	86 µmol/l
HbA1c	45 mmol/mol (6.3%)

Urine dipstick shows no proteinuria. Her blood pressure today in clinic is 134/76 mmHg.

What is the most appropriate course of action?

○ Add gliclazide ×

○ Increase amlodipine ×

○ Increase ramipril ×

○ Add losartan ×

○ No changes to medication required ×

Submit answer

Reference ranges ⌄

Score: **21.3%**

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Question 128 of 129 ✓

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K^+	4.4 mmol/l
Urea	7.2 mmol/l
Creatinine	86 μmol/l
HbA1c	45 mmol/mol (6.3%)

Urine dipstick shows no proteinuria. Her blood pressure today in clinic is 134/76 mmHg.

What is the most appropriate course of action?

Add gliclazide	2%
Increase amlodipine	5%
Increase ramipril	4%
Add losartan	1%
No changes to medication required	89%

Newly diagnosed patient of black African or African-Caribbean origin with hypertension - add a calcium channel blocker

Important for me Less important

Her diabetic control is good - NICE do not advocate changing treatment at this stage unless the HbA1c is >= 6.5%.

As she has no complications from her diabetes the blood pressure target is < 140/80 mmHg. No changes are therefore required to her antihypertensive regime.

👍 👎 Discuss (7) Improve

Next question >

Hypertension: management ★

NICE published updated guidelines for the management of hypertension in 2019. Some of the key changes include:

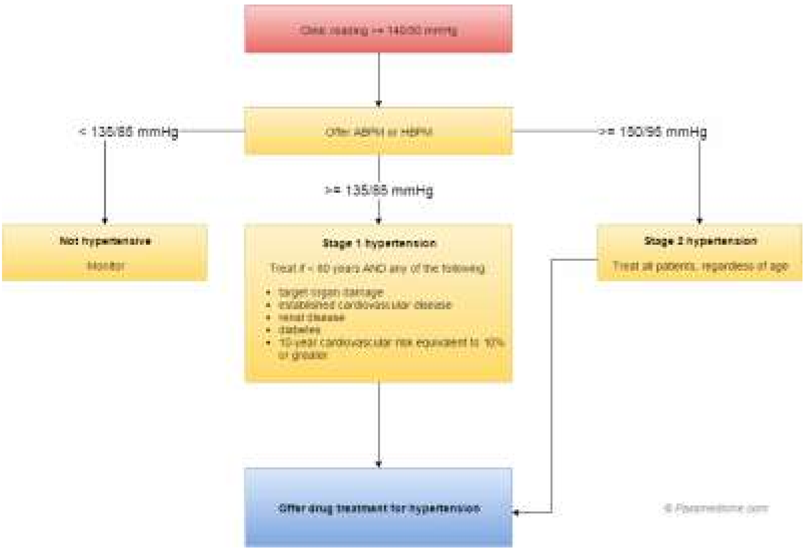
- lowering the threshold for treating stage 1 hypertension in patients < 80 years from 20% to 10%
- angiotensin receptor blockers can be used instead of ACE-inhibitors where indicated
- if a patient is already taking an ACE-inhibitor or angiotensin receptor blocker, then a calcium channel blocker OR a thiazide-like diuretic can be used. Previously only a calcium channel blocker was recommended

Blood pressure classification

This becomes relevant later in some of the management decisions that NICE advocates.

Stage	Criteria
Stage 1 hypertension	Clinic BP >= 140/90 mmHg and subsequent ABPM daytime average or HBPM average BP >= 135/85 mmHg

Stage	Criteria
Stage 2 hypertension	Clinic BP >= 160/100 mmHg and subsequent ABPM daytime average or HBPM average BP >= 150/95 mmHg
Severe hypertension	Clinic systolic BP >= 180 mmHg, or clinic diastolic BP >= 120 mmHg


Clinic reading >= 140/90 mmHg
Offer ABPM or HBPM
< 135/85 mmHg
>= 150/95 mmHg
>= 135/85 mmHg
Not hypertensive
Monitor
Stage 1 hypertension
Treat if < 80 years AND any of the following
target organ damage
established cardiovascular disease
renal disease
diabetes
10-year cardiovascular risk equivalent to 10% or greater
Stage 2 hypertension
Treat all patients, regardless of age
Offer drug treatment for hypertension
© Passmedicine.com

Flow chart showing simplified schematic for diagnosis hypertension following NICE guidelines

Managing hypertension

Lifestyle advice should not be forgotten and is frequently tested in exams:

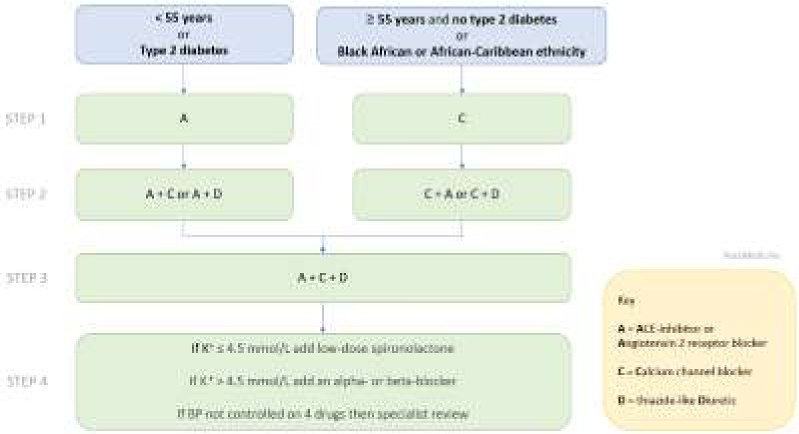
- a low salt diet is recommended, aiming for less than 6g/day, ideally 3g/day. The average adult in the UK consumes around 8-12g/day of salt. A recent BMJ paper* showed that lowering salt intake can have a significant effect on blood pressure. For example, reducing salt intake by 6g/day can lower systolic blood pressure by 10mmHg
- caffeine intake should be reduced
- the other general bits of advice remain: stop smoking, drink less alcohol, eat a balanced diet rich in fruit and vegetables, exercise more, lose weight

ABPM/HBPM >= 135/85 mmHg (i.e. stage 1 hypertension)

- treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 10% or greater
- in 2019, NICE made a further recommendation, suggesting that we should '*consider antihypertensive drug treatment in addition to lifestyle advice for adults aged under 60 with stage 1 hypertension and an estimated 10-year risk below 10%.* '. This seems to be due to evidence that QRISK may underestimate the lifetime probability of developing cardiovascular disease

ABPM/HBPM >= 150/95 mmHg (i.e. stage 2 hypertension)
- offer drug treatment regardless of age

For patients < 40 years consider specialist referral to exclude secondary causes.


< 55 years
or
Type 2 diabetes
≥ 55 years and no type 2 diabetes
or
Black African or African-Caribbean ethnicity
STEP 1
A
C
STEP 2
A + C or A + D
C + A or C + D
STEP 3
A + C + D
STEP 4
If K⁺ ≤ 4.5 mmol/L add low-dose spironolactone
If K⁺ > 4.5 mmol/L add an alpha- or beta-blocker
If BP not controlled on 4 drugs then specialist review
Key
A = ACE-inhibitor or Angiotensin 2 receptor blocker
C = Calcium channel blocker
D = thiazide-like Diuretic

Flow chart showing the management of hypertension as per current NICE guideliness

Step 1 treatment
- patients < 55-years-old or a background of type 2 diabetes mellitus: **A**CE inhibitor or a **A**ngiotensin receptor blocker (**A**CE-i or **A**RB): **(A)**
  - angiotensin receptor blockers should be used where ACE inhibitors are not tolerated (e.g. due to a cough)
- patients >= 55-years-old or of black African or African-Caribbean origin: **C**alcium channel blocker **(C)**
  - ACE inhibitors have reduced efficacy in patients of black African or African-Caribbean origin are therefore not used first-line

Step 2 treatment

- if already taking an **A**CE-i or **A**RB add a **C**alcium channel blocker or a thiazide-like **D**iuretic
- if already taking a **C**alcium channel blocker add an **A**CE-i or **A**RB or a thiazide-like **D**iuretic
    - for patients of black African or African-Caribbean origin taking a calcium channel blocker for hypertension, if they require a second agent consider an angiotensin receptor blocker in preference to an ACE inhibitor
- **(A + C) or (A + D)** or **(C + A) or (C + D)**

Step 3 treatment

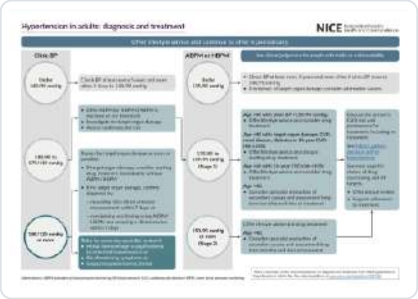
- add a third drug to make, i.e.:
    - if already taking an **(A + C)** then add a **D**
    - if already **(A + D)** then add a **C**
- **(A + C + D)**

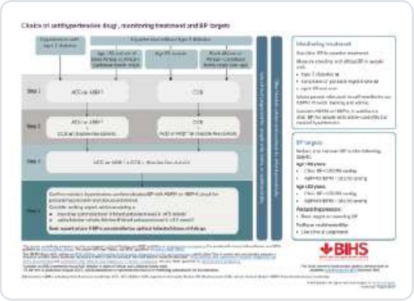
Step 4 treatment

- NICE define step 4 as resistant hypertension and suggest either adding a 4th drug (as below) or seeking specialist advice
- first, check for:
    - confirm elevated clinic BP with ABPM or HBPM
    - assess for postural hypotension.
    - discuss adherence
- if potassium < 4.5 mmol/l add low-dose spironolactone
- if potassium > 4.5 mmol/l add an alpha- or beta-blocker

Patients who fail to respond to step 4 measures should be referred to a specialist. NICE recommend:

> *If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.*





Blood pressure targets

	Clinic BP	**ABPM / HBPM**
Age < 80 years	140/90 mmHg	135/85 mmHg
Age > 80 years	150/90 mmHg	145/85 mmHg

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Links

NICE

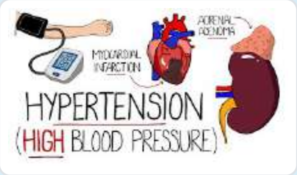
👍 162 👎 162

2019 Hypertension guidelines

Suggest link

Report broken link

Media


ADRENAL ADENOMA
MYOCARDIAL INFARCTION
HYPERTENSION
(HIGH BLOOD PRESSURE)

Hypertension Explained

Rhesus Medicine - YouTube 👍 2 👎 0

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Score: **21.9%**

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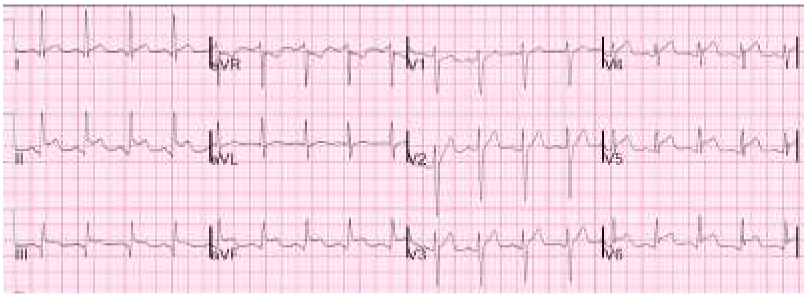
A 45-year-old man is referred to the ambulatory care team with a 4-day history of left-sided chest pain and dyspnoea. He describes the chest pain as feeling sharp in nature and is associated with occasional shortness of breath and palpitations, particularly on exertion. He reports that his daughter has just recovered from scarlet fever and he has also been affected by coryzal symptoms over the past 2 weeks. His past medical history includes hypertension for which he takes ramipril 10mg OD.

An ECG is taken, as shown below:



© Image used on license from Dr Smith, University of Minnesota

Given the likely diagnosis, what is the most appropriate management?

- ○ Calcium gluconate ×
- ○ Colchicine ×
- ○ Dual antiplatelet therapy ×
- ○ Intravenous broad-spectrum antibiotics ×
- ○ Simple analgesia and reassurance ×

Submit answer

Reference ranges

A 45-year-old man is referred to the ambulatory care team with a 4-day history of left-sided chest pain and dyspnoea. He describes the chest pain as feeling sharp in nature and is associated with occasional shortness of breath and palpitations, particularly on exertion. He reports that his daughter has just recovered from scarlet fever and he has also been affected by coryzal symptoms over the past 2 weeks. His past medical history includes hypertension for which he takes ramipril 10mg OD.

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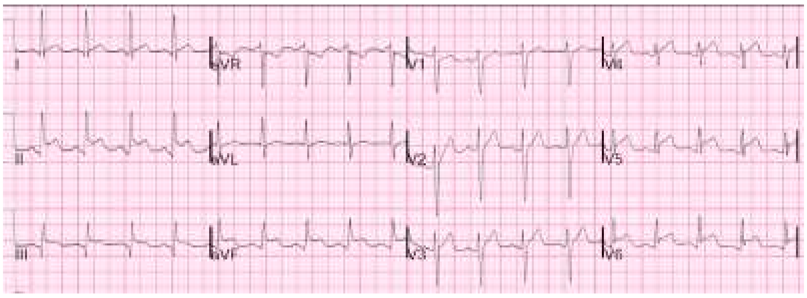

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Given the likely diagnosis, what is the most appropriate management?

Calcium gluconate	2%
Colchicine	67%
Dual antiplatelet therapy	9%
Intravenous broad-spectrum antibiotics	2%
Simple analgesia and reassurance	20%

Colchicine is correct. This patient has a likely diagnosis of pericarditis given the history of pleuritic chest pain after a recent coryzal illness. This patient's ECG shows widespread ST elevation with PR depression, both of which are characteristic signs of pericarditis. The treatment of pericarditis is with NSAIDs and colchicine, particularly for idiopathic or viral pericarditis.

Calcium gluconate is incorrect. ECG findings consistent with hyperkalaemia include tall tented T waves, PR interval prolongation and QRS widening. It does not cause widespread ST elevation or PR depression, as shown here.

Dual antiplatelet therapy is incorrect. Although this patient's ECG does demonstrate ST elevation, the ST elevation is widespread and not specific to one coronary artery territory. Furthermore, the nature of the ST depression is concave and is associated with PR depression, making pericarditis much more likely.

Intravenous broad-spectrum antibiotics is incorrect. This would be indicated for infective endocarditis which is a condition that typically presents with fevers, constitutional symptoms (e.g. malaise, weight loss, night sweats) and fatigue as the predominant features, in contrast to pleuritic chest pain that is being described here. Infective endocarditis does not explain this patient's ECG findings.

Simple analgesia and reassurance is incorrect. This would be indicated for musculoskeletal chest pain or perhaps even costochondritis which is characterised by pleuritic chest pain that may follow a recent viral illness. However, unlike pericarditis, costochondritis is the inflammation of the costochondral joints and does not produce ECG changes, as seen in this presentation.

👍 👎 Discuss (4) Improve

Acute pericarditis

Acute pericarditis is a condition referring to inflammation of the pericardial sac, lasting for less than 4-6 weeks.

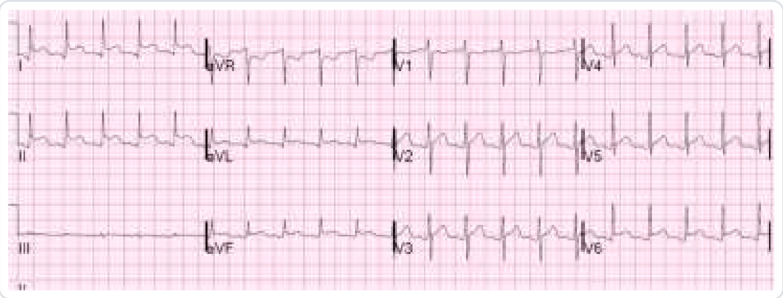
Aetiology

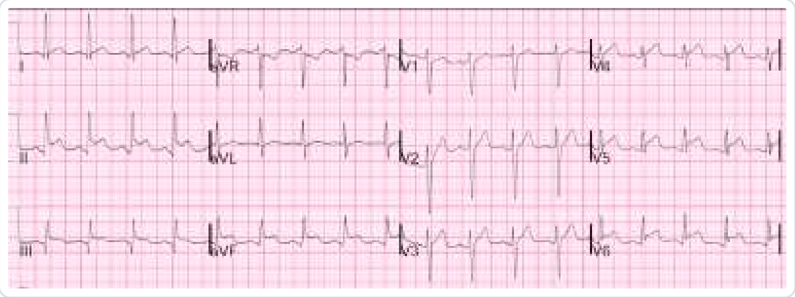
- viral infections (Coxsackie)
- tuberculosis
- uraemia
- post-myocardial infarction
    - early (1-3 days): fibrinous pericarditis

    - late (weeks to months): autoimmune pericarditis (Dressler's syndrome)
- radiotherapy
- connective tissue disease
    - systemic lupus erythematosus
    - rheumatoid arthritis
- hypothyroidism
- malignancy
    - lung cancer
    - breast cancer
- trauma

Features
- chest pain: may be pleuritic. Is often relieved by sitting forwards
- other symptoms include a non-productive cough, dyspnoea and flu-like symptoms
- pericardial rub

Investigations
- ECG changes
    - the changes in pericarditis are often global/widespread, as opposed to the 'territories' seen in ischaemic events
    - 'saddle-shaped' ST elevation
    - PR depression: most specific ECG marker for pericarditis
- all patients with suspected acute pericarditis should have transthoracic echocardiography
- bloods
    - inflammatory markers
    - troponin: around 30% of patients may have an elevated troponin - this indicates possible myopericarditis





Management

- the majority of patients can be managed as outpatients
    - patients who have high-risk features such as fever > 38°C or elevated troponin should be managed as an inpatient
- treat any underlying cause
    - most patients however will have pericarditis secondary to viral infection, meaning no specific treatment is indicated
- strenuous physical activity should be avoided until symptom resolution and normalisation of inflammatory markers
- a combination of NSAIDs and colchicine is now generally used for first-line for patients with acute idiopathic or viral pericarditis
    - until symptom resolution and normalisation of inflammatory markers (usually 1-2 weeks) followed by tapering of dose recommended over

123

Textbooks

High-yield textbook

Extended textbook

Links

Life in the fast lane

👍 36 👎 15

Pericarditis review

Suggest link
Report broken link

Media

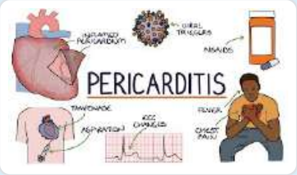
PERICARDITIS & PERICARDIAL EFFUSIONS
OSMOSIS.org

Pericarditis and pericardial effusions

Osmosis - YouTube 👍 2 👎 0

Pericardial rub - REQUIRES HEADPHONES

Thinklabs - YouTube 👍 2 👎 0


PERICARDITIS
NSAIDS
FEVER
CHEST PAIN

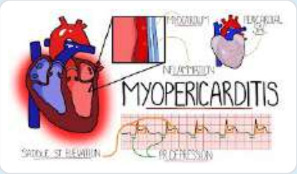
Pericarditis

Zero To Finals - YouTube 👍 0 👎 0

PERICARDITIS

Pericarditis

Armando Hasudungan - YouTube 👍 2 👎 1


MYOCARDIUM
PERICARDIAL
MYOPERICARDITIS
SADDLE ST ELEVATION
PR DEPRESSION

Myopericarditis Explained - Myocarditis & Pericarditis

Rhesus Medicine - YouTube 👍 1 👎 2

Report broken media

Score: **21.7%**

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